Day : Tuesday
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Inventor Name Search Result

Your Search was:

Last Name = STEPHAN
First Name = JEAN-PHILIPPE

Application#	Patent#	Status	Date Filed	Title	Inventor Name
10243189	Not Issued	030	09/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE
10081056	Not Issued	168	02/20/2002	COMPOSITIONS AND METHODS FOR THE DIAGNOSIS AND TREATMENT OF DISORDERS INVOLVING ANGIOGENESIS	STEPHAN, JEAN-PHILIPPE F.
10119480	Not Issued	030	04/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216159	Not Issued	020	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216160	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216162	Not Issued	041	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216163	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING	STEPHAN, JEAN-PHILIPPE F.

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10216164	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216165	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216166	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216167	Not Issued	020	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10216168	Not Issued	030	08/09/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10218631	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10218784	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10218849	Not Issued	020	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10218930	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10218956	Not	030	08/12/2002	SECRETED AND	STEPHAN, JEAN-

	Issued			TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	PHILIPPE F.
10219003	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219010	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219060	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219061	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219062	Not Issued	030	08/12/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219063	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219064	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219065	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219066	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND	STEPHAN, JEAN-PHILIPPE F.

				NUCLEIC ACIDS ENCODING THE SAME	
10219067	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219068	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219069	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219071	Not Issued	020	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219072	Not Issued	020	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219073	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219074	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219075	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219076	Not Issued	031	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.

10219077	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219150	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219464	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219465	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219466	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219467	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219468	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219469	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219470	Not Issued	030	08/14/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219471	Not Issued	020	08/14/2002	SECRETED AND TRANSMEMBRANE	STEPHAN, JEAN-PHILIPPE F.

				POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	
10219472	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219473	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219474	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219475	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.
10219478	Not Issued	030	08/13/2002	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	STEPHAN, JEAN-PHILIPPE F.

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15/AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0015168704 BIOSIS NO.: 200500075769
Pathogenesis of tuberous sclerosis subependymal giant cell astrocytomas: Biallelic inactivation of TSC1 or TSC2 leads to mTOR activation
AUTHOR: Chan Jennifer A; Zhang Hongbing; Roberts Penelope S; Jozwiak Sergiusz; Wieslawa Grajkowska; Lewin-Kowalik Joanna; Kotulska Katarzyna; Kwiatkowski David J (Reprint)
AUTHOR ADDRESS: Dept Med, Brigham and Womens Hosp, 1 Blackfan Circle,CHNRB6-216, Boston, MA, 02115, USA**USA
AUTHOR E-MAIL ADDRESS: dk@rics.bwh.harvard.edu
JOURNAL: Journal of Neuropathology & Experimental Neurology 63 (12): p 1236-1242 December 2004 2004
MEDIUM: print
ISSN: 0022-3069 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In the central nervous system, tuberous sclerosis complex (TSC) is characterized by a range of lesions including cortical tubers, white matter heterotopias, subependymal nodules, and subependymal giant cell astrocytomas (SEGAs), Recent studies have implicated an important role for the TSC genes TSC1 and TSC2, in a signaling pathway involving the mammalian target of rapamycin (mTOR) kinase. We performed immunohistochemical and genetic analyses on SEGAs from 7 TSC patients, 4 with mutations in TSC1, and 3 with mutations in TSC2. SEGA cells show high levels of phospho-S6K, phospho-S6, and phospho-Stat3, all proteins downstream of and indicative of mTOR activation. Such expression is not seen in histologically normal control tissue. Five of 6 SEGAs also showed evidence of biallelic mutation of TSC1 or TSC2, suggesting that SEGAs develop due to complete loss of a functional tuberin-hamartin complex. We conclude that TSC SEGAs likely arise through a two-hit mechanism of biallelic inactivation of TSC1 or TSC2, leading to activation of the mTOR kinase.

15/AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013971079 BIOSIS NO.: 200200564590
Association between a high-expressing interferon-gamma allele and a lower frequency of kidney angiomyolipomas in TSC2 patients
AUTHOR: Dabora Sandra L (Reprint); Roberts Penelope; Nieto Andres; Perez Ron; Jozwiak Sergiusz; Franz David; Bissler John; Thiele Elizabeth A; Sims Katherine; Kwiatkowski David J
AUTHOR ADDRESS: Division of Hematology, Brigham and Women's Hospital, 221 Longwood Avenue, LMRC 301, Boston, MA, 02115, USA**USA
JOURNAL: American Journal of Human Genetics 71 (4): p750-758 October, 2002 2002
MEDIUM: print
ISSN: 0002-9297
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Tuberous sclerosis complex (TSC) is a familial hamartoma syndrome in which renal involvement is common and, at times, life threatening. We have investigated the potential effect of a non-TSC gene on renal disease in a cohort of 172 TSC patients with TSC2 mutations. Patients were genotyped for an interferon-gamma (IFN-gamma) microsatellite polymorphism, within intron 1, for which one common allele (allele 2, with 12 CA repeats) has been shown to have a higher expression of IFN-gamma. A chi2 analysis was used to examine the association between IFN-gamma allele 2 and the development of kidney angiomyolipomas (KAMLs) in this TSC2 cohort. Because of the age-dependent development of KAMLs in TSC, we initially focused on the 127 patients who were >5 years old. Additional subgroup analyses were done to investigate the influence of age and gender. The transmission/disequilibrium test (TDT) was also performed in a subset of this cohort (46 probands) for whom parent and/or sibling samples were available for analysis. Both chi2 analysis and TDT suggested an association between IFN-gamma allele 2 and the absence of KAMLs in patients who have known TSC2 mutations. Among the 127 patients who were >5 years old, KAMLs were present in 95 (75%) and were absent in 32 (25%). In the group with KAML present, the frequency of IFN-gamma allele 2 was 56%; in the group with KAML absent, the frequency of IFN-gamma allele 2 was significantly higher, at 78% (P = .02, by chi2 analysis). The family-based TDT analysis gave similar results, with a TDT statistic (TDT chi2 = 5.45) corresponding to a P value of .02. Subgroup analyses show that both age and gender may influence the impact of this association. Although these results should be replicated in other populations with TSC, the present study suggests that modifier genes play a role in the variable expression of TSC and also suggests a potential therapy for KAMLs in patients with TSC.

15/AB/3 (Item 3 from file: 5)

0013919517 BIOSIS NO.: 200200513028

Human pancreatic epithelial progenitor cells and methods of isolation and use thereof

AUTHOR: Roberts Penelope E (Reprint); Mather Jennie Powell
AUTHOR ADDRESS: Millbrae, CA, USA**USA

JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1261 (3): Aug. 20, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6436704 PATENT DATE GRANTED: August 20, 2002 20020820
PATENT CLASSIFICATION: 435-366 PATENT ASSIGNEE: Raven Biotechnologies, Inc. PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The invention discloses a substantially pure population of human pancreatic progenitor cells and methods of isolating and culturing the pancreatic progenitor cells. By carefully manipulating the microenvironment of the pancreatic progenitor cells, multiple passages are attainable wherein the pancreatic progenitor cells do not senesce and furthermore, are capable of becoming functional exocrine or endocrine cells. In addition, several methods of use of human pancreatic progenitor cells are disclosed herein.

15/AB/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013870174 BIOSIS NO.: 200200463685
SNP identification, haplotype analysis, and parental origin of mutations in TSC2
AUTHOR: Roberts Penelope S; Chung Joon; Jozwiak Sergiusz; Dabora Sandra L; Franz David N; Thiele Elizabeth A; Kwiatkowski David J (Reprint)
AUTHOR ADDRESS: Hematology Division, Brigham and Women's Hospital, Harvard Medical School, 221 Longwood Ave., Boston, MA, 02115, USA**USA
JOURNAL: Human Genetics 111 (1): p96-101 July, 2002 2002
MEDIUM: print
ISSN: 0340-6717
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inactivating mutations in the TSC2 gene, consisting of 41coding exons in 40 kb on 16p13, cause the hamartoma syndrome tuberous sclerosis. During TSC2 mutational analysis we identified ten SNPs that occur within or close to exon boundaries at minor allele frequencies greater than 5%. We determined the haplotypes for six of these SNPs and the microsatellite marker kg8 in the 3' region of TSC2 in a set of 40 parent-child trios. The most common haplotypes accounted for 53%, 11%, 6%, and 5% of chromosomes. Thirty-eight TSC2 mutation-bearing haplotypes had a similar distribution, indicating that there was no haplotype that predisposed to mutation in this region of TSC2. Family analysis was possible in 12 sporadic cases, and indicated that the mother was the parent of origin in 7 cases (3 point mutations, 2 small deletions, 2 large deletions), while the father was in 5 cases (2 point mutations, 3 small deletions). We conclude that TSC2 mutations occur at substantial frequency on both the maternally and paternally derived TSC2 alleles, in contrast to many other genetic diseases including NF1. The observations have implications for genetic counseling in TSC.

15/AB/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012897713 BIOSIS NO.: 200100069552
Mutational analysis in a cohort of 224 tuberous sclerosis patients indicates increased severity of TSC2, compared with TSC1, disease in multiple organs
AUTHOR: Dabora Sandra L (Reprint); Jozwiak Sergiusz; Franz David Neal; Roberts Penelope S; Nieto Andres; Chung Joon; Choy Yew-Sing; Reeve Mary Pat; Thiele Elizabeth; Egelhoff John C; Kasprzyk-Obara Jolanta; Domanska-Pakiela Dorota; Kwiatkowski David J (Reprint)
AUTHOR ADDRESS: Genetics Laboratory, Division of Hematology, Brigham and Women's Hospital, 221 Longwood Avenue, LMRC 301, Boston, MA, 02115, USA** USA
JOURNAL: American Journal of Human Genetics 68 (1): p64-80 January, 2001 2001
MEDIUM: print
ISSN: 0002-9297
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Tuberous sclerosis (TSC) is a relatively common hamartoma syndrome caused by mutations in either of two genes, TSC1 and TSC2. Here we report comprehensive mutation analysis in 224 index patients with TSC and correlate mutation findings with clinical features. Denaturing high-performance liquid chromatography, long-range polymerase chain reaction (PCR), and quantitative PCR were used for mutation detection. Mutations were identified in 186 (83%) of 224 of cases, comprising 138 small TSC2 mutations, 20 large TSC2 mutations, and 28 small TSC1 mutations. A standardized clinical assessment instrument covering 16 TSC manifestations was used. Sporadic patients with TSC1 mutations had, on average, milder disease in comparison with patients with TSC2 mutations, despite being of similar age. They had a lower frequency of seizures and moderate-to-severe mental retardation, fewer subependymal nodules and cortical tubers, less-severe kidney involvement, no retinal hamartomas, and less-severe facial angiofibroma. Patients in whom no mutation was found also had disease that was milder, on average, than that in patients with TSC2 mutations and was somewhat distinct from patients with TSC1 mutations. Although there was overlap in the spectrum of many clinical features of patients with TSC1 versus TSC2 mutations, some features (grade 2-4 kidney cysts or angiomyolipomas, forehead plaques, retinal hamartomas, and liver angiomyolipomas) were very rare or not seen at all in TSC1 patients. Thus both germline and somatic mutations appear to be less common in TSC1 than in TSC2. The reduced severity of disease in patients without defined mutations suggests that many of these patients are mosaic for a TSC2 mutation and/or have TSC because of mutations in an as-yet-unidentified locus with a relatively mild clinical phenotype.

15/AB/6 (Item 6 from file: 5)

0012342648 BIOSIS NO.: 200000060961

Selective cloning of cell surface proteins involved in organ development: Epithelial glycoprotein is involved in normal epithelial differentiation

AUTHOR: Stephan Jean-Philippe (Reprint); Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Research Immunochemistry-AAT, Genentech, Inc., South San Francisco, CA, USA**USA

JOURNAL: Endocrinology 140 (12): p5841-5854 Dec., 1999 1999
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Coordinating the activities of neighboring cells during development in multicellular organisms requires complex cellular interactions involving secreted, cell surface, and extracellular matrix components. Although most cloning efforts have concentrated on secreted molecules, recent work has emphasized the importance of membrane-bound molecules during development. To identify developmental genes, we raised antibodies to normal embryonic pancreatic epithelial cell surface proteins. These antibodies were characterized and used to clone the genes coding for the proteins by a panning strategy. Using this approach, we cloned the rat homologue of the mouse epithelial glycoprotein (EGP). Our immunohistochemistry data, describing the expression of EGP during rat development, as well as our in vitro data, looking at the effect of the

anti-EGP antibody and the extracellular domain of EGP on embryonic pancreatic epithelial cell number and volume, strongly suggest a role for EGP during pancreatic development.

15/AB/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012209803 BIOSIS NO.: 199900469463
Distribution and function of the adhesion molecule BEN during rat development
AUTHOR: Stephan Jean-Philippe (Reprint); Bald Laura; Roberts Penelope E (Reprint); Lee James; Gu Qimin; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Department of Protein Chemistry, Genentech, Inc., South San Francisco, CA, 94080-4990, USA**USA
JOURNAL: Developmental Biology 212 (2): p264-277 Aug. 15, 1999 1999
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It is well established that the notochord influences the development of adjacent neural and mesodermal tissue. Involvement of the notochord in the differentiation of the dorsal pancreas has been demonstrated. However, our knowledge of the signals involved in pancreatic development is still incomplete. In order to identify proteins potentially implicated during pancreatic differentiation, we raised and characterized monoclonal antibodies against previously established embryonic pancreatic ductal epithelial cell lines (BUD and RED). Using the MAb 2117, the cell surface antigen 2117 (Ag 2117) was cloned. The predicted sequence for Ag 2117 is the rat homologue of BEN. Initially reported as a protein expressed on epithelial cells of the chicken bursa of Fabricius, BEN is expressed in a variety of tissues during development and described as a marker for the developing central and peripheral chicken nervous systems. A role has been suggested for BEN in the adhesion of stem cells and progenitor cells to the blood-forming tissue microenvironment. In this study, we demonstrate that BEN, initially expressed exclusively in the notochord during the early development of rat, is implicated in pancreatic development. We show that Ag 2117 regulates the pancreatic epithelial cell growth through the ras and Jun kinase pathways. In addition, we demonstrate that Ag 2117 is able to regulate the expression of the transcription factor PDX1, required for insulin gene expression, in embryonic pancreas organ cultures.

15/AB/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011769119 BIOSIS NO.: 199900028779
Biological response to ErbB ligands in nontransformed cell lines correlates with a specific patterns of receptor expression
AUTHOR: Sundaresan Srividya (Reprint); Roberts Penelope E; King Kathleen L; Sliwkowski Mark X; Mather Jennie P
AUTHOR ADDRESS: 1 DNA Way, MS 45, Genentech Inc., S. San Frnacisco, CA 94080, USA**USA
JOURNAL: Endocrinology 139 (12): p4756-4764 Dec., 1998 1998
MEDIUM: print

ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The human epidermal growth factor receptor (HER or ErbB) family consists of four distinct members, including the epidermal growth factor (EGF) receptor (EGFR, HER1, or ErbB1), ErbB2 (HER2 or neu), ErbB3 (HER3), and ErbB4 (HER4). Activation ofthese receptors plays an important role in the regulation of cell proliferation, differentiation, and survival in several different tissues. Binding of a specific ligand to one of the ErbB receptors triggers the formation of specific receptor homo- and heterodimers, with ErbB2 being the preferred signaling partner. We analyzed the levels of various ErbB receptor messenger RNAs in a series of nontransformed cell lines by real time quantitative RT-PCR. The cell lines chosen were derived from a variety of tissues, including pancreas, lung, heart, and nervous system. Further, we measured biological responses in these cell lines upon treatment with EGF, betacellulin, and two types of neuregulins, heregulin and sensory and motor neuron-derived factor. All cell lines examined expressed detectable levels of ErbB2. High levels of expression of ErbB3 were correlated with responveness to heregulin and sensory and motor neuron-derived factor, whereas high levels of EGFR expression were correlated with responsiveness to EGF and betacellulin. Moreover, the sensitivity of a cell line to ErbB ligands was also correlated with the levels of expression of the appropriate ErbB receptors in that cell line. These results are consistent with our hypothesis that appropriate biological responsiveness to ErbB ligands is determined by the levels of expression of specific ErbB receptor combinations within a given tissue.

15/AB/9 (Item 9 from file: 5)

0011759356 BIOSIS NO.: 199900019016

Selective cloning of cell surface proteins involved in organ development: EGP is involved in normal epithelial differentiation

AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Helmrich Angela; Barnes David; Devaux Brigitte; Mather Jennie P

JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p436A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

15/AB/10 (Item 10 from file: 5)

0011756285 BIOSIS NO.: 199900015945

Distribution and function of the rat homologue of the adhesion molecule BEN during development

AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qumin; Devaux Brigitte; Mather Jennie P

AUTHOR ADDRESS: Dep. Protein Chem., Genentech Inc., South San Francisco, CA 94080-4990, USA**USA
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p200A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

15/AB/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008881321 BIOSIS NO.: 199396045737
Follistatin modulates activin activity in a cell- and tissue-specific manner
AUTHOR: Mather Jennie P (Reprint); Roberts Penelope E; Krummen Lynne A
AUTHOR ADDRESS: Cell Culture R and D, Genentech, Inc., 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrinology 132 (6): p2732-2734 1993
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The high affinity activin-binding protein, follistatin, has recently been shown to block activin-stimulated activities in several in vitro systems. In the present study we sought to extend these observations and investigate the effects of follistatin on the activity of activin in stimulating the re-aggregation of Sertoli cell monolayers and proliferation of testicular germ cells, as measured by incorporation of (3H)-thymidine in vitro. Germ-Sertoli cell cocultures prepared from 21 day old rats were treated with media alone or media containing recombinant human (rh) activin A or rh activin B with or without follistatin, the low affinity activin-binding protein, alpha-2 macroglobulin, or a monoclonal antibody (mAB) known to block activin B activity. Follistatin blocked the ability of activin A to stimulate reaggregation of Sertoli cell monolayers when present at a 2-fold ratio (wt/wt) to activin. However, in these same cultures, follistatin bad no effect on the ability of activin A to stimulate (3H)-thymidine incorporation. In activin B-treated cultures, both responses could be blocked by the addition of a neutralizing mAB directed against activin B. These results suggest that follistatin can modulate activin action in a cell-type specific fashion, and that this protein may play an important role in regulating the bioavailability of activin.

15/AB/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008777454 BIOSIS NO.: 199395079720
Childhood cancer and paternal exposure to ionizing radiation: Preliminary findings from the Oxford Survey of Childhood Cancers
AUTHOR: Sorahan Tom (Reprint); Roberts Penelope J
AUTHOR ADDRESS: Cancer Epidemiol. Res. Unit, Dep. Public Health Epidemiol.,

University Birmingham, Edgbaston, Birmingham B15 2TT, UK**UK
JOURNAL: American Journal of Industrial Medicine 23 (2): p343-354 1993
ISSN: 0271-3586
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Paternal occupational data already collected as part of the Oxford Survey of Childhood Cancers have been reviewed. Information on occupations during or before the relevant pregnancy was sought for 15,279 children dying from cancer in England, Wales, and Scotland (UK) in the period 1953-81, and for an equal number of matched controls. Estimates were made for paternal exposure to human-made external ionizing radiation in the six months before conception of the survery child sbd as judged from job histories and dates of birth. Assessments were also made for potential exposure to unsealed sources of radionuclides. Of the eight fathers placed in the highest dose group (gtoreq 10 mSv, external radiation), four were cases and four were controls. For the second dose group (5-9 mSv), the corresponding numbers were eight and four, and for the lowest exposed group (1-4 mSv), they were 55 and 42. There were 27 case fathers with potential exposure to radionuclides and only 10 control fathers. The independent effects of the two radiation variables were assessed by means of multiple logistic regression. Relative risks for estimated doses of external radiation were close to unity, but for radionuclide exposure the relative risk was 2.87 (95% CI = 1.15-7.13). These preliminary findings suggest that paternal exposure to radionuclides is a more likely risk factor for childhood cancer than exposure to external radiation.

15/AB/13 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

15086182 PMID: 14641237
A 34 bp deletion within TSC2 is a rare polymorphism, not a pathogenic mutation.
Roberts Penelope S; Ramesh Vijaya; Dabora Sandra; Kwiatkowski David J
Hematology Division, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
Annals of human genetics (England) Nov 2003, 67 (Pt 6) p495-503, ISSN 0003-4800 Journal Code: 0416661
Contract/Grant No.: 24279; PHS; 31535; PHS
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Tuberous sclerosis (TSC) is an autosomal dominant hamartoma syndrome due to mutations in either TSC1 or TSC2. Previous reports have identified a mutation consisting of a 34 bp deletion affecting portions of exon 38 and the adjacent intron 38 of TSC2. We found this genetic variation in 4 of 800 TSC patients screened for mutations in TSC1 and TSC2. In every case, the variant was present in one unaffected parent of the sporadically affected TSC child. By RT-PCR analysis of RNA samples from two additional families with this genetic variant, we demonstrate that the allele with the deletion generates about 50% normal RNA transcript, and 50% RNA transcript including intron 38. In addition, there is no correlation between the extent of splicing and clinical status of family members. We also excluded the possibility of mosaicism in the parents with this variant. We conclude that

this deletion is a rare polymorphism that does not cause TSC, but may be a modifier of the TSC phenotype.
?

Day : Tuesday
Date: 3/15/2005

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Time: 14:49:24

Inventor Name Search Result

Your Search was:

Last Name = ROBERTS
First Name = PENELOPE

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09546577	6436704	150	04/10/2000	HUMAN PANCREATIC EPITHELIAL PROGENITOR CELLS AND METHODS OF ISOLATION AND USE THEREOF	ROBERTS, PENELOPE E.
09614483	Not Issued	161	07/10/2000	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	ROBERTS, PENELOPE E.
10119601	Not Issued	041	04/09/2002	HUMAN PANCREATIC EPITHELIAL PROGENITOR CELLS AND METHODS OF ISOLATION AND USE THEREOF	ROBERTS, PENELOPE E.
10600802	Not Issued	030	06/19/2003	NOVEL RAAG10 CELL SURFACE TARGET AND A FAMILY OF ANTIBODIES RECOGNIZING THAT TARGET	ROBERTS, PENELOPE E.
10672878	Not Issued	071	09/26/2003	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	ROBERTS, PENELOPE E.
60362867	Not Issued	159	03/07/2002	ANTI-EPCAM ANTIBODY MPA6	ROBERTS, PENELOPE E.
60390203	Not Issued	159	06/19/2002	NOVEL RAAG10 CELL SURFACE TARGET EXPRESSED IN CANCER AND A FAMILY ANTIBODIES RECOGNIZING THAT TARGET	ROBERTS, PENELOPE E.
60578103	Not	018	06/07/2004	LUCA31 AND ANTIBODIES	ROBERTS,

	Issued			THAT BIND THERETO	PENELOPE E.
60578105	Not Issued	020	06/07/2004	LUCA9 AND ANTIBODIES THAT BIND THERETO	ROBERTS, PENELOPE E.
60649007	Not Issued	020	01/31/2005	LUCA2 AND ANTIBODIES THAT BIND THERETO	ROBERTS, PENELOPE E.
60649979	Not Issued	020	02/03/2005	ANTIBODIES TO ONCOSTATIN M RECEPTOR	ROBERTS, PENELOPE E.
07479130	Not Issued	166	02/09/1990	LUNG CELL LINE AND METHODS OF USE	ROBERTS, PENELOPE E.
07910260	5830685	150	07/16/1992	A METHOD OF PRODUCING PROTEINS USING MAMMALIAN LUNG CELL LINES	ROBERTS, PENELOPE E.
07919994	Not Issued	166	07/27/1992	LUNG CELL LINE AND METHODS OF USE	ROBERTS, PENELOPE E.
08060466	5364785	150	05/11/1993	A METHOD OF ISOLATING LUNG CELL LINE	ROBERTS, PENELOPE E.
08455755	5736345	150	05/31/1995	AN ASSAY FOR GROWTH PROMOTING FACTORS UTILIZING MAMMALIAN LUNG CELL LINES	ROBERTS, PENELOPE E.
09218539	Not Issued	161	12/22/1998	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	ROBERTS, PENELOPE E.

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** roberts **First Name** penelope Search

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Day : Tuesday
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Inventor Name Search Result

Your Search was:

Last Name = BALD
First Name = LAURA

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09545659	Not Issued	095	04/10/2000	HUMAN OVARIAN MESOTHELIAL CELLS AND METHODS OF ISOLATION AND USES THEREOF	BALD, LAURA
10744508	Not Issued	094	12/22/2003	HUMAN OVARIAN MESOTHELIAL CELLS AND METHODS OF ISOLATION AND USES THEREOF	BALD, LAURA
09614483	Not Issued	161	07/10/2000	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	BALD, LAURA N.
10445179	Not Issued	095	05/23/2003	HUMAN OVARIAN MESOTHELIAL CELLS AND METHODS OF ISOLATION AND USES THEREOF	BALD, LAURA N.
10672878	Not Issued	071	09/26/2003	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	BALD, LAURA N.
60577896	Not Issued	020	06/07/2004	TES4 AND ANTIBODIES THAT BIND THERETO	BALD, LAURA N.
09218539	Not Issued	161	12/22/1998	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	BALD, LAURA N.
10853037	Not Issued	020	05/25/2004	CONTENT CUSTOMIZATION WITH RESIZABILITY AND CONTEXT-SENSITIVITY	BALDWIN, LAURA
60623519	Not Issued	020	10/29/2004	GEOGRAPHIC LOCATION AND SEARCH NEAR ME	BALDWIN, LAURA J.

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** bald **First Name** laura Search

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13/AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013091764 BIOSIS NO.: 200100263603
fucosyltransferase1 and H-type complex carbohydrates modulate epithelial cell proliferation during prostatic branching morphogenesis
AUTHOR: Marker Paul C; Stephan Jean-Philippe; Lee James; Bald Laura; Mather Jennie P; Cunha Gerald R (Reprint)
AUTHOR ADDRESS: Department of Anatomy, University of California San Francisco, San Francisco, CA, 94143, USA**USA
JOURNAL: Developmental Biology 233 (1): p95-108 May 1, 2001 2001
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The prostate undergoes branching morphogenesis dependent on paracrine interactions between the prostatic epithelium and the urogenital mesenchyme. To identify cell-surface molecules that function in this process, monoclonal antibodies raised against epithelial cell-surface antigens were screened for antigen expression in the developing prostate and for their ability to alter development of prostates grown in serum-free organ culture. One antibody defined a unique expression pattern in the developing prostate and inhibited growth and ductal branching of cultured prostates by inhibiting epithelial cell proliferation. Expression cloning showed that this antibody binds fucosyltransferase1, an alpha-(1,2)-fucosyltransferase that synthesizes H-type structures on the complex carbohydrate modifications of some proteins and lipids. The lectin UEA I that binds H-type 2 carbohydrates also inhibited development of cultured prostates. These data demonstrate a previously unrecognized role for fucosyltransferase1 and H-type carbohydrates in controlling the spatial distribution of epithelial cell proliferation during prostatic branching morphogenesis. We also show that fucosyltransferase1 is expressed by epithelial cells derived from benign prostatic hyperplasia or prostate cancer; thus, fucosyltransferase1 may also contribute to pathological prostatic growth. These data further suggest that rare individuals who lack fucosyltransferase1 (Bombay phenotype) should be investigated for altered reproductive function and/or altered susceptibility to benign prostatic hyperplasia and prostate cancer.

13/AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012342648 BIOSIS NO.: 200000060961
Selective cloning of cell surface proteins involved in organ development: Epithelial glycoprotein is involved in normal epithelial differentiation
AUTHOR: Stephan Jean-Philippe (Reprint); Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Research Immunochemistry-AAT, Genentech, Inc., South San Francisco, CA, USA**USA
JOURNAL: Endocrinology 140 (12): p5841-5854 Dec., 1999 1999
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Coordinating the activities of neighboring cells during development in multicellular organisms requires complex cellular interactions involving secreted, cell surface, and extracellular matrix components. Although most cloning efforts have concentrated on secreted molecules, recent work has emphasized the importance of membrane-bound molecules during development. To identify developmental genes, we raised antibodies to normal embryonic pancreatic epithelial cell surface proteins. These antibodies were characterized and used to clone the genes coding for the proteins by a panning strategy. Using this approach, we cloned the rat homologue of the mouse epithelial glycoprotein (EGP). Our immunohistochemistry data, describing the expression of EGP during rat development, as well as our in vitro data, looking at the effect of the anti-EGP antibody and the extracellular domain of EGP on embryonic pancreatic epithelial cell number and volume, strongly suggest a role for EGP during pancreatic development.

13/AB/3 (Item 3 from file: 5)

0012209803 BIOSIS NO.: 199900469463

Distribution and function of the adhesion molecule BEN during rat development

AUTHOR: Stephan Jean-Philippe (Reprint); Bald Laura; Roberts Penelope E (Reprint); Lee James; Gu Qimin; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Department of Protein Chemistry, Genentech, Inc., South San Francisco, CA, 94080-4990, USA**USA

JOURNAL: Developmental Biology 212 (2): p264-277 Aug. 15, 1999 1999
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It is well established that the notochord influences the development of adjacent neural and mesodermal tissue. Involvement of the notochord in the differentiation of the dorsal pancreas has been demonstrated. However, our knowledge of the signals involved in pancreatic development is still incomplete. In order to identify proteins potentially implicated during pancreatic differentiation, we raised and characterized monoclonal antibodies against previously established embryonic pancreatic ductal epithelial cell lines (BUD and RED). Using the MAb 2117, the cell surface antigen 2117 (Ag 2117) was cloned. The predicted sequence for Ag 2117 is the rat homologue of BEN. Initially reported as a protein expressed on epithelial cells of the chicken bursa of Fabricius, BEN is expressed in a variety of tissues during development and described as a marker for the developing central and peripheral chicken nervous systems. A role has been suggested for BEN in the adhesion of stem cells and progenitor cells to the blood-forming tissue microenvironment. In this study, we demonstrate that BEN, initially expressed exclusively in the notochord during the early development of rat, is implicated in pancreatic development. We show that Ag 2117 regulates the pancreatic epithelial cell growth through the ras and Jun kinase pathways. In addition, we demonstrate that Ag 2117 is able to regulate the expression of the transcription factor PDX1, required for insulin gene expression, in embryonic pancreas organ cultures.

13/AB/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011968248 BIOSIS NO.: 199900227908
Vaccination with the extracellular domain of p185neu prevents mammary tumor development in neu transgenic mice
AUTHOR: Esserman Laura J; Lopez Theresa; Montes Ruben; Bald Laura N; Fendly Brian M; Campbell Michael J (Reprint)
AUTHOR ADDRESS: Department of Surgery, UCSF/Mount Zion Medical Center, 1600 Divisadero, Rm C-342, San Francisco, CA, 94120, USA**USA
JOURNAL: Cancer Immunology Immunotherapy 47 (6): p337-342 Feb., 1999 1999
MEDIUM: print
ISSN: 0340-7004
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The HER2/neu oncogene product, p185HER2/neu, is overexpressed on the surface of many human breast cancers. Strains of transgenic mice have been developed that express the rat neu oncogene in mammary epithelial cells and develop spontaneous mammary tumors that overexpress p185neu. This model provides an ideal system for testing interventions to prevent tumor development. In this study, we immunized neu-transgenic mice with a vaccine consisting of the extracellular domain of p185neu (NeuECD). Immunized mice developed Neu-specific humoral immune responses, as measured by circulating anti-Neu antibodies in their sera, and cellular immune responses, as measured by lymphocyte proliferation to NeuECD in vitro. In addition, the subsequent development of mammary tumors was significantly lower in immunized mice than in controls and vaccine treatment was associated with a significant increase in median survival.

13/AB/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011759356 BIOSIS NO.: 199900019016
Selective cloning of cell surface proteins involved in organ development: EGP is involved in normal epithelial differentiation
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Helmrich Angela; Barnes David; Devaux Brigitte; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p436A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/AB/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011756285 BIOSIS NO.: 199900015945
Distribution and function of the rat homologue of the adhesion molecule BEN

during development
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qumin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Dep. Protein Chem., Genentech Inc., South San Francisco, CA 94080-4990, USA**USA
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p200A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

13/AB/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011227083 BIOSIS NO.: 199800021330
Characterization of cell surface proteins using antibodies raised to antigens from pancreatic cell lines
AUTHOR: Stephan Jean-Philippe; Bald Laura; Roberts Penny; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 8 (SUPPL.): p328A Nov., 1997 1997
MEDIUM: print
CONFERENCE/MEETING: 37th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 13-17, 1997; 19971213
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

13/AB/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010303102 BIOSIS NO.: 199698770935
Analysis of heregulin-induced ErbB2 phosphorylation with a high-throughput kinase receptor activation enzyme-linked immunosorbent assay
AUTHOR: Sadick Michael D (Reprint); Sliwkowski Mark X; Nuijens Andrew; Bald Laura; Chiang Nancy; Lofgren Julie A; Wong Wai Lee T
AUTHOR ADDRESS: Dep. BioAnalytical Technology, Res. Immunochemistry, 460 Pt. San Bruno Boulevard, South San Francisco, CA 94080, USA**USA
JOURNAL: Analytical Biochemistry 235 (2): p207-214 1996 1996
ISSN: 0003-2697
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A rapid, sensitive, and high-throughput assay has been developed to quantify ligand-induced receptor tyrosine kinase activation in terms of receptor phosphorylation. The assay, termed a kinase receptor activation enzyme-linked immunosorbant assay (KIRA-ELISA), consists of two separate microtiter plates, one for cell culture, ligand stimulation, and cell lysis/receptor solubilization and the other plate for receptor capture and phosphotyrosine ELISA. The assay was developed for analysis of heregulin-induced ErbB2 activation and utilizes the stimulation of

intact receptor on the adherent breast carcinoma cell line, MCF-7. Membrane proteins are solubilized via Triton X-100 lysis and the receptor is captured in ELISA wells coated with ErbB2-specific antibodies with no cross-reaction to ErbB3 or ErbB4. The degree of receptor phosphorylation is then quantified by antiphosphotyrosine ELISA. A reproducible standard curve is generated with a EC-50 of approximately 360 pm for heregulin beta-1-177-244 (HRG-BETA-1-177-244). When identical samples of hrg-BETA-1-177-244 are analyzed by both the KIRA-ELISA and quantitative antiphosphotyrosine Western blot analysis, the results correlate very closely with one another. The assay described in this report is able to specifically quantify tyrosine phosphorylation of ErbB2 that results from the interaction of HRG with ErbB3 and/or ErbB4.

13/AB/9 (Item 9 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0008982351 BIOSIS NO.: 199497003636

Monoclonal antibody based ELISAs for measurement of activins in biological fluids

AUTHOR: Wong Wai Lee T (Reprint); Garg Shaily J; Woodruff Teresa; Bald Laura; Fendly Brian; Lofgren James A

AUTHOR ADDRESS: Genentech Inc., 460 Pt. San Bruno Blvd., So. San Francisco, CA 94080, USA**USA

JOURNAL: Journal of Immunological Methods 165 (1): p1-10 1993 1993

ISSN: 0022-1759

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Two sensitive monoclonal antibody (MAb)-based enzyme-linked immunosorbent assays (ELISAs), one for activin A (homodimer of beta-A subunits) and one for activin B (homodimer of beta-B subunits) in plasma have been developed. The activin A ELISA had an effective range of 0.2-50 ng/ml while the activin B ELISA's range was 0.1-25 ng/ml in human serum. Both ELISAs were specific with lt 0.01% cross-reactivity with related hormones and follistatin (an activin binding protein), however the presence of recombinant human follistatin caused a decrease in measured level of activin A and B spiked human samples. The assay was linear across the standard curve range with intra- and interassay coefficients of variation were less than 15%. The level of activins in female serum range from 0.3 to 10.4 ng/ml. In summary, we have developed a reliable, convenient and rapid MAb-based enzyme immunoassay for determination of activin A and B levels in human serum which are also applicable for buffer, mouse and monkey serum matrices. This assay will be useful for studying the regulation and role of activin A and B in health and disease.

13/AB/10 (Item 10 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0008862496 BIOSIS NO.: 199396026912

Development of a specific and sensitive two-site enzyme linked immunosorbent assay for measurement of inhibin A in serum

AUTHOR: Baly Deborah L (Reprint); Allison David E; Krummen Lynne A; Woodruff Teresa K; Soules Michael R; Chen Sharon A; Fendly Brian M; Bald Laura N; Mather Jennie P; Lucas Catherine

AUTHOR ADDRESS: Genetech, Inc., 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrinology 132 (5): p2099-2108 1993
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A polyclonal chicken antiserum against purified 32-kilodalton (kDa) recombinant inhibin-A (rh-InhA) and two monoclonal antibodies (mAb) against either rh-InhA (11B5) or 28-kDa recombinant activin-A (rh-ActA; 9A9) were used to develop three sensitive InhA enzyme-linked immunosorbent assays (ELISAs). The sensitivity of an ELISA using affinity-purified chicken anti-rh-InhA (Ck) for both coat and capture (Ck/Ck) averaged 78 +- 3 pg/ml, while the mAb/Ck ELISAs (11B5/Ck or 9A9/Ck) averaged 100 +- 6 pg/ml in a 10% serum matrix, with intra- and interassay coefficients of variation of 2-5% and 8-10%, respectively, for all assays. The ELISA formats did not cross-react with purified rh-ActA or recombinant human transforming growth factor-b-1 or detect any immunoreactive proteins in medium conditioned by cell lines expressing rh-ActA or recombinant human transforming growth factor-beta-1. The Ck/Ck ELISA detected significant amounts of immunoreactivity in medium from cells expressing the free alpha-subunit of inhibin and recombinant inhibin-B (rh-InhB). In contrast, the mAb/Ck ELISAs showed no cross-reactivity to medium conditioned by these two cell lines. All three ELISA formats detected rh-InhA added to either human or rat serum in vitro or serum from rats injected with rh-InhA. The Ck/Ck and 9A9/Ck ELISAs successfully quantitated inhibin in sera from patients undergoing ovulation induction and in rats (with or without sc administration of pregnant female serum gonadotropin). The 11B5/Ck ELISA appeared to be specific for the 32-kDa form of inhibin, while the 9A9/Ck ELISA was useful in quantitating inhibin-A in biological fluids, with little cross-reactivity to free alpha-chain or inhibin-B.
?

Day : Tuesday
Date: 3/15/2005

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Time: 14:48:01

Inventor Name Search Result

Your Search was:

Last Name = MATHER
First Name = JENNIE

Application#	Patent#	Status	Date Filed	Title	Inventor Name
08548345	Not Issued	001	11/01/1995	NORMAL NEURAL EPITHELIAL PRECURSOR CELLS	MATHER, JENNIE
60035194	Not Issued	159	11/01/1995	NORMAL NEURAL EPITHELIAL PRECURSOR CELLS	MATHER, JENNIE
09614483	Not Issued	161	07/10/2000	COMPOSITIONS AND METHODS FOR GENERATING MONOCLONAL ANTIBODIES REPRESENTATIVE OF A SPECIFIC CELL TYPE	MATHER, JENNIE P.
09660590	6653287	150	09/13/2000	USE OF LEUKEMIA INHIBITORY FACTOR AND ENDOTHELIN ANTAGONISTS	MATHER, JENNIE P.
09665350	Not Issued	041	09/18/2000	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEUIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09699119	Not Issued	161	10/27/2000	RECEPTOR ACTIVATION BY GAS6	MATHER, JENNIE P.
09728342	6399331	150	11/30/2000	METHOD FOR CULTURING RECOMBINANT CELLS	MATHER, JENNIE P.
09902572	Not Issued	041	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902615	Not Issued	061	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

09902634	Not Issued	041	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902654	Not Issued	161	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902692	Not Issued	161	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902736	Not Issued	094	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902759	Not Issued	161	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902775	6686451	150	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902853	Not Issued	161	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09902903	Not Issued	161	07/10/2001	ANTI-PRO293 ANTIBODIES	MATHER, JENNIE P.
09902979	Not Issued	161	07/10/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903520	Not Issued	041	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

09903562	Not Issued	092	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903640	Not Issued	041	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903663	Not Issued	168	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903749	Not Issued	041	07/11/2001	ANTI-PRO211 POLYPEPTIDE ANTIBODIES	MATHER, JENNIE P.
09903786	Not Issued	161	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903806	Not Issued	161	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903823	Not Issued	161	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903910	Not Issued	168	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903925	Not Issued	120	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09903943	Not Issued	161	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

09904011	Not Issued	120	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904119	Not Issued	161	07/11/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCONDING THE SAME	MATHER, JENNIE P.
09904485	Not Issued	120	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904553	Not Issued	161	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904658	Not Issued	168	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904766	Not Issued	061	07/12/2001	PRO269 POLYPEPTIDES	MATHER, JENNIE P.
09904786	Not Issued	041	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904805	Not Issued	161	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904820	Not Issued	161	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904838	Not Issued	041	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

09904859	Not Issued	161	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904877	Not Issued	061	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904938	Not Issued	120	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904956	Not Issued	041	07/14/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09904992	Not Issued	041	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09905056	Not Issued	061	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09905075	Not Issued	041	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09905088	Not Issued	161	07/12/2001	PRO293 POLYPEPTIDE	MATHER, JENNIE P.
09905106	Not Issued	168	07/13/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.
09905125	6664376	150	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

09905291	Not Issued	161	07/12/2001	SECRETED AND TRANSMEMBRANE POLYPEPTIDES AND NUCLEIC ACIDS ENCODING THE SAME	MATHER, JENNIE P.

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TYPE S11/MEDIUM, AB/1-51
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11/AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0014887184 BIOSIS NO.: 200400257941
Secreted and transmembrane polypeptides and nucleic acids encoding the same
AUTHOR: Ashkenazi Avi (Reprint); Botstein David; Desnoyers Luc; Eaton Dan L ; Ferrara Napoleone; Filvaroff Ellen; Fong Sherman; Gao Wei-Qiang; Gerber Hanspeter; Gerritsen Mary E; Goddard Audrey; Godowski Paul J; Grimaldi J Christopher; Gurney Austin L; Hillan Kenneth J; Kljavin Ivar J; Mather Jennie P; Pan James; Paoni Nicholas F; Roy Margaret Ann; Stewart Timothy A; Tumas Daniel; Williams P Mickey; Wood William I
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1281 (3): Apr. 20, 2004 2004
MEDIUM: e-file
PATENT NUMBER: US 6723535 PATENT DATE GRANTED: April 20, 2004 20040420
PATENT CLASSIFICATION: 435-691 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for producing the polypeptides of the present invention.

11/AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2005 BIOSIS. All rts. reserv.

0014761167 BIOSIS NO.: 200400141924
Secreted and transmembrane polypeptides and nucleic acids encoding the same
AUTHOR: Desnoyers Luc (Reprint); Goddard Audrey; Godowski Paul J; Gurney Austin L; Mather Jennie P; Williams P Mickey; Wood William I
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1279 (1): Feb. 3, 2004 2004
MEDIUM: e-file
PATENT NUMBER: US 6686451 PATENT DATE GRANTED: February 03, 2004 20040203
PATENT CLASSIFICATION: 530-3871 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present invention is directed to novel polypeptides and to nucleic acid molecules encoding those polypeptides. Also provided herein are vectors and host cells comprising those nucleic acid sequences, chimeric polypeptide molecules comprising the polypeptides of the present invention fused to heterologous polypeptide sequences, antibodies which bind to the polypeptides of the present invention and to methods for

producing the polypeptides of the present invention.

11/AB/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014674540 BIOSIS NO.: 200400045297
Use of leukemia inhibitory factor and endothelin antagonists
AUTHOR: Ferrara Napoleone (Reprint); King Kathleen; Luis Elizabeth; Mather Jennie P; Paoni Nicholas F
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1276 (4): Nov. 25, 2003 2003
MEDIUM: e-file
PATENT NUMBER: US 6653287 PATENT DATE GRANTED: November 25, 2003 20031125
PATENT CLASSIFICATION: 514-21 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A leukemia inhibitory factor antagonist, alone or in combination with an endothelin antagonist, may be used for treatment of heart failure. The antagonist(s) are administered in a chronic fashion, in therapeutically effective amounts, to achieve this purpose.

11/AB/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013991015 BIOSIS NO.: 200200584526
Receptor polypeptides and their production and uses
AUTHOR: Cox Edward T (Reprint); Mather Jennie P; Sliwkowski Mary B; Woodruff Teresa K
AUTHOR ADDRESS: Foster City, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1262 (4): Sep. 24, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6455262 PATENT DATE GRANTED: September 24, 2002 20020924
PATENT CLASSIFICATION: 435-71 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: An isolated TGF-beta supergene family (TSF) receptor polypeptide is provided. This polypeptide preferably is an inhibin/activin receptor polypeptide and has at least 75% sequence identity with the mature human inhibin/activin receptor sequence. Also provided is a method for purifying TGF-beta supergene family members such as inhibin or activin using the polypeptide, and a method for screening for compounds with TGF-beta supergene family member activity by contacting the compound with the polypeptide and detecting if binding has occurred and the compound is active.

11/AB/5 (Item 5 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)
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0013919517 BIOSIS NO.: 200200513028
Human pancreatic epithelial progenitor cells and methods of isolation and use thereof
AUTHOR: Roberts Penelope E (Reprint); Mather Jennie Powell
AUTHOR ADDRESS: Millbrae, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1261 (3): Aug. 20, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6436704 PATENT DATE GRANTED: August 20, 2002 20020820
PATENT CLASSIFICATION: 435-366 PATENT ASSIGNEE: Raven Biotechnologies, Inc. PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The invention discloses a substantially pure population of human pancreatic progenitor cells and methods of isolating and culturing the pancreatic progenitor cells. By carefully manipulating the microenvironment of the pancreatic progenitor cells, multiple passages are attainable wherein the pancreatic progenitor cells do not senesce and furthermore, are capable of becoming functional exocrine or endocrine cells. In addition, several methods of use of human pancreatic progenitor cells are disclosed herein.

11/AB/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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0013853524 BIOSIS NO.: 200200447035
Human Mullerian duct-derived epithelial cells and methods of isolation and uses thereof
AUTHOR: Li Rong-hao (Reprint); Mather Jennie Powell
AUTHOR ADDRESS: LaJolla, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1260 (2): July 9, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6416999 PATENT DATE GRANTED: July 09, 2002 20020709
PATENT CLASSIFICATION: 435-366 PATENT ASSIGNEE: Raven Biotechnologies, Inc., South San Francisco, CA, USA PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This invention discloses a substantially pure population of human Mullerian duct-derived epithelial cells and methods of isolating and culturing the Mullerian duct-derived epithelial cells. By carefully manipulating the microenvironment in which the Mullerian duct-derived epithelial cells are grown, multiple passages are attainable wherein the Muillerian duct-derived epithelial cells are capable of becoming uterine, cervical, vaginal, and oviductal cells. In addition, several uses of human Mullerian duct-derived epithelial cells and cells differentiating therefrom are disclosed herein.

11/AB/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013818643 BIOSIS NO.: 200200412154
Cell arrays and the uses thereof
AUTHOR: Li Ronghao (Reprint); Mather Jennie P
AUTHOR ADDRESS: La Jolla, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1259 (3): June 18, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6406840 PATENT DATE GRANTED: June 18, 2002 20020618
PATENT CLASSIFICATION: 435-13 PATENT ASSIGNEE: bioMosaic Systems, Inc., South San Francisco, CA, USA PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The present invention provides cell arrays comprising a plurality of tubes containing populations of cells that are immobilized therein. The arrays are particularly useful for conducting comparative cell-based analyses. Specifically, the subject arrays allow protein-protein interactions to be studied in multiple types of cell simultaneously. The arrays also support simultaneous detection of the differential expression of a target polynucleotide in a multiplicity of cell types derived from multiple subjects. The subject arrays further permit high throughput screening for candidate modulators of a signal transduction pathway of interest. Further provided by the invention are kits, computer-implemented methods and systems for conducting the comparative cell-based analyses.

11/AB/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013784410 BIOSIS NO.: 200200377921
Method for culturing recombinant cells
AUTHOR: Mather Jennie P (Reprint); Ullrich Axel
AUTHOR ADDRESS: Millbrae, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1259 (1): June 4, 2002 2002
MEDIUM: e-file
PATENT NUMBER: US 6399331 PATENT DATE GRANTED: June 04, 2002 20020604
PATENT CLASSIFICATION: 435-691 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A method for culturing a recombinant host cell comprising: determining a polypeptide factor for a polypeptide factor-dependent host cell; transforming said host cell with nucleic acid encoding said polypeptide factor; transforming the host cell with nucleic acid encoding a desired protein; and, culturing the transformed host cells in a medium lacking the polypeptide factor.

11/AB/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013118378 BIOSIS NO.: 200100290217
Mer receptor activation by gas6
AUTHOR: Chen Jian; Hammonds R Glenn (Reprint); Godowski Paul J; Mark Melanie R; Mather Jennie P; Li Ronghao
AUTHOR ADDRESS: Berkeley, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1242 (1): Jan. 2, 2001 2001
MEDIUM: e-file
PATENT NUMBER: US 6169070 PATENT DATE GRANTED: January 02, 2001 20010102
PATENT CLASSIFICATION: 514-2 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The translation product of the growth arrest-specific gene 6 (gas6) has been identified as an activator of the Mer receptor protein tyrosine kinase. The invention accordingly provides methods of activating Mer receptor in cells expressing it by exposing them to exogenous gas6 polypeptides. Also provided are methods of enhancing the growth, differentiation, or survival of such cells using gas6 polypeptides.

11/AB/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013091764 BIOSIS NO.: 200100263603
fucosyltransferase1 and H-type complex carbohydrates modulate epithelial cell proliferation during prostatic branching morphogenesis
AUTHOR: Marker Paul C; Stephan Jean-Philippe; Lee James; Bald Laura; Mather Jennie P; Cunha Gerald R (Reprint)
AUTHOR ADDRESS: Department of Anatomy, University of California San Francisco, San Francisco, CA, 94143, USA**USA
JOURNAL: Developmental Biology 233 (1): p95-108 May 1, 2001 2001
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The prostate undergoes branching morphogenesis dependent on paracrine interactions between the prostatic epithelium and the urogenital mesenchyme. To identify cell-surface molecules that function in this process, monoclonal antibodies raised against epithelial cell-surface antigens were screened for antigen expression in the developing prostate and for their ability to alter development of prostates grown in serum-free organ culture. One antibody defined a unique expression pattern in the developing prostate and inhibited growth and ductal branching of cultured prostates by inhibiting epithelial cell proliferation. Expression cloning showed that this antibody binds fucosyltransferase1, an alpha-(1,2)-fucosyltransferase that synthesizes H-type structures on the complex carbohydrate modifications of some proteins and lipids. The lectin UEA I that binds H-type 2 carbohydrates also inhibited development of cultured prostates. These data demonstrate

a previously unrecognized role for fucosyltransferase1 and H-type carbohydrates in controlling the spatial distribution of epithelial cell proliferation during prostatic branching morphogenesis. We also show that fucosyltransferase1 is expressed by epithelial cells derived from benign prostatic hyperplasia or prostate cancer; thus, fucosyltransferase1 may also contribute to pathological prostatic growth. These data further suggest that rare individuals who lack fucosyltransferase1 (Bombay phenotype) should be investigated for altered reproductive function and/or altered susceptibility to benign prostatic hyperplasia and prostate cancer.

11/AB/11 (Item 11 from file: 5)

0012988463 BIOSIS NO.: 200100160302

Differentiation of granulosa cell line: Follicle-stimulating hormone induces formation of lamellipodia and filopodia via the adenylyl cyclase/cyclic adenosine monophosphate signal

AUTHOR: Grieshaber Nicole A; Boitano Scott; Ji Inhae; Mather Jennie P; Ji Tae H (Reprint)
AUTHOR ADDRESS: Department of Chemistry, University of Kentucky, Lexington, KY, 40506-0055, USA**USA

JOURNAL: Endocrinology 141 (9): p3461-3470 September, 2000 2000
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: FSH plays a crucial role in granulosa cell differentiation and follicular development during the ovulation cycle. The early events of granulosa cell differentiation in cell culture involve changes in the cell morphology and cell-to-cell interactions. To determine the cause and signaling mechanism for these changes, we examined an undifferentiated rat ovarian granulosa cell line that grows in a defined serum-free medium, expresses the FSH receptor, terminally differentiates when exposed to FSH, and undergoes apoptosis upon FSH withdrawal. FSH bound the FSH receptor on rat ovarian granulosa cells, and the liganded receptor activated adenylyl cyclase (AC) to produce cAMP but did not mobilize Ca2+. In addition, we observed massive reorganization of the actin cytoskeleton within 3 h of FSH treatment. This involves formation of lamellipodia and filopodia and spreading of multilayer cell aggregates to monolayers. This actin reorganization and cell transformation could also be induced by the AC activator, forskolin, in the absence of FSH. Furthermore, AC inhibitors blocked the FSH-dependent actin reorganization and transformation. On the other hand, phospholipase C inhibitors did not block the FSH-induced changes. Taken together, our observations indicate that the AC/cAMP signal is necessary and sufficient for FSH-dependent granulosa cell differentiation, including massive reorganization of the actin cytoskeleton and changes in the cell morphology and cell-to-cell interactions. There is no evidence that the phospholipase C signal and Ca2+ mobilization are involved in this process.

11/AB/12 (Item 12 from file: 5)

0012683448 BIOSIS NO.: 200000401761
Method of treating a nervous system injury with cultured schwann cells
AUTHOR: Mather Jennie P (Reprint); Li Ronghao; Chen Jian
AUTHOR ADDRESS: Millbrae, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1232 (1): Mar. 7, 2000 2000
MEDIUM: e-file
PATENT NUMBER: US 6033660 PATENT DATE GRANTED: March 07, 2000 20000307
PATENT CLASSIFICATION: 424-937 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A method for enhancing the survival and/or proliferation of Schwann cells (especially human Schwann cells) in cell culture is disclosed which involves culturing the cells in serum free culture medium comprising gas6 and other mitogenic agents, such as heregulin and forskolin. The culturing step is generally preceded by a pre-incubation period wherein nerve tissue comprising the Schwann cells is cultured under appropriate conditions and for a period of time such that demyelination occurs. The isolated Schwann cells can be used as cellular prostheses to treat patients with nervous system injuries. The invention also provides a cell culture medium for culturing Schwann cells.

11/AB/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012457172 BIOSIS NO.: 200000175485
Identification and regulation of receptor tyrosine kinases Rse and Mer and their ligand Gas6 in testicular somatic cells
AUTHOR: Chan Michael CW (Reprint); Mather Jennie P; McCray Glynis; Lee Will M
AUTHOR ADDRESS: Department of Zoology, The University of Hong Kong, Pokfulam Rd, Hong Kong, China**China
JOURNAL: Journal of Andrology 21 (2): p291-302 March-April, 2000 2000
MEDIUM: print
ISSN: 0196-3635
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Receptor tyrosine kinases act to convey extracellular signals to intracellular signaling pathways and ultimately control cell proliferation and differentiation. Rse, Axl, and Mer belong to a newly identified family of cell adhesion molecule-related receptor tyrosine kinase. They bind the vitamin K-dependent protein growth arrest-specific gene 6 (Gas6), which is also structurally related to the anticoagulation factor Protein S. The aim of this study is to investigate the possible role of Rse/Axl/Mer tyrosine kinase receptors and their ligand in regulating testicular functions. Gene expression of Rse, Axl, Mer, and Gas6 in the testis was studied by reverse transcriptase-polymerase chain reaction (RT-PCR) and Northern blot analysis. The results indicated that receptors Rse and Mer and the ligand Gas6 were expressed in the rat endothelial cell line (TR1), mouse Leydig cell line (TM3), rat peritubular myoid cell line (TRM), mouse Sertoli cell line (TM4), and primary rat Sertoli cells. Axl was not expressed in the testicular

somatic cells by RT-PCR or Northern blot analysis. The highest level of expression of Gas6 messenger RNA (mRNA) was observed in the Sertoli cells, and its expression was responsive to the addition of forskolin in vitro. The effects of serum, insulin, and transferrin on Gas6 expression by TM4 cells were examined. It was shown that they all exhibited an up-regulating effect on Gas6 expression. The forskolin-stimulated Gas6 expression was accompanied by an increase in tyrosine phosphorylation of the Rse receptor in vitro, suggesting that Gas6 may exhibit an autocrine effect in the Sertoli cells through multiple tyrosine kinase receptors. Our studies so far have demonstrated that tyrosine kinase receptors Rse and Mer and their ligand Gas6 are widely expressed in the testicular somatic cell lines and may play a marked role in promoting testicular cell survival.

11/AB/14 (Item 14 from file: 5)

0012342648 BIOSIS NO.: 200000060961

Selective cloning of cell surface proteins involved in organ development: Epithelial glycoprotein is involved in normal epithelial differentiation

AUTHOR: Stephan Jean-Philippe (Reprint); Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Research Immunochemistry-AAT, Genentech, Inc., South San Francisco, CA, USA**USA

JOURNAL: Endocrinology 140 (12): p5841-5854 Dec., 1999 1999
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Coordinating the activities of neighboring cells during development in multicellular organisms requires complex cellular interactions involving secreted, cell surface, and extracellular matrix components. Although most cloning efforts have concentrated on secreted molecules, recent work has emphasized the importance of membrane-bound molecules during development. To identify developmental genes, we raised antibodies to normal embryonic pancreatic epithelial cell surface proteins. These antibodies were characterized and used to clone the genes coding for the proteins by a panning strategy. Using this approach, we cloned the rat homologue of the mouse epithelial glycoprotein (EGP). Our immunohistochemistry data, describing the expression of EGP during rat development, as well as our in vitro data, looking at the effect of the anti-EGP antibody and the extracellular domain of EGP on embryonic pancreatic epithelial cell number and volume, strongly suggest a role for EGP during pancreatic development.

11/AB/15 (Item 15 from file: 5)

0012283116 BIOSIS NO.: 200000001429

Rse receptor activation

AUTHOR: Chen Jian (Reprint); Hammonds Glenn R; Godowski Paul J; Mark Melanie R; Mather Jennie P; Li Ronghao
AUTHOR ADDRESS: Burlingame, CA, USA**USA

JOURNAL: Official Gazette of the United States Patent and Trademark Office

Patents 1226 (3): Sep. 21, 1999 1999
MEDIUM: print
PATENT NUMBER: US 5955420 PATENT DATE GRANTED: Sep. 21, 1999 19990921
PATENT CLASSIFICATION: 514-2 PATENT ASSIGNEE: Genentech, Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Citation
LANGUAGE: English

11/AB/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012209803 BIOSIS NO.: 199900469463
Distribution and function of the adhesion molecule BEN during rat development
AUTHOR: Stephan Jean-Philippe (Reprint); Bald Laura; Roberts Penelope E (Reprint); Lee James; Gu Qimin; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Department of Protein Chemistry, Genentech, Inc., South San Francisco, CA, 94080-4990, USA**USA
JOURNAL: Developmental Biology 212 (2): p264-277 Aug. 15, 1999 1999
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It is well established that the notochord influences the development of adjacent neural and mesodermal tissue. Involvement of the notochord in the differentiation of the dorsal pancreas has been demonstrated. However, our knowledge of the signals involved in pancreatic development is still incomplete. In order to identify proteins potentially implicated during pancreatic differentiation, we raised and characterized monoclonal antibodies against previously established embryonic pancreatic ductal epithelial cell lines (BUD and RED). Using the MAb 2117, the cell surface antigen 2117 (Ag 2117) was cloned. The predicted sequence for Ag 2117 is the rat homologue of BEN. Initially reported as a protein expressed on epithelial cells of the chicken bursa of Fabricius, BEN is expressed in a variety of tissues during development and described as a marker for the developing central and peripheral chicken nervous systems. A role has been suggested for BEN in the adhesion of stem cells and progenitor cells to the blood-forming tissue microenvironment. In this study, we demonstrate that BEN, initially expressed exclusively in the notochord during the early development of rat, is implicated in pancreatic development. We show that Ag 2117 regulates the pancreatic epithelial cell growth through the ras and Jun kinase pathways. In addition, we demonstrate that Ag 2117 is able to regulate the expression of the transcription factor PDX1, required for insulin gene expression, in embryonic pancreas organ cultures.

11/AB/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011769119 BIOSIS NO.: 199900028779
Biological response to ErbB ligands in nontransformed cell lines correlates with a specific patterns of receptor expression

AUTHOR: Sundaresan Srividya (Reprint); Roberts Penelope E; King Kathleen L; Sliwkowski Mark X; Mather Jennie P
AUTHOR ADDRESS: 1 DNA Way, MS 45, Genentech Inc., S. San Frnacisco, CA 94080, USA**USA
JOURNAL: Endocrinology 139 (12): p4756-4764 Dec., 1998 1998
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The human epidermal growth factor receptor (HER or ErbB) family consists of four distinct members, including the epidermal growth factor (EGF) receptor (EGFR, HER1, or ErbB1), ErbB2 (HER2 or neu), ErbB3 (HER3), and ErbB4 (HER4). Activation ofthese receptors plays an important role in the regulation of cell proliferation, differentiation, and survival in several different tissues. Binding of a specific ligand to one of the ErbB receptors triggers the formation of specific receptor homo- and heterodimers, with ErbB2 being the preferred signaling partner. We analyzed the levels of various ErbB receptor messenger RNAs in a series of nontransformed cell lines by real time quantitative RT-PCR. The cell lines chosen were derived from a variety of tissues, including pancreas, lung, heart, and nervous system. Further, we measured biological responses in these cell lines upon treatment with EGF, betacellulin, and two types of neuregulins, heregulin and sensory and motor neuron-derived factor. All cell lines examined expressed detectable levels of ErbB2. High levels of expression of ErbB3 were correlated with responveness to heregulin and sensory and motor neuron-derived factor, whereas high levels of EGFR expression were correlated with responsiveness to EGF and betacellulin. Moreover, the sensitivity of a cell line to ErbB ligands was also correlated with the levels of expression of the appropriate ErbB receptors in that cell line. These results are consistent with our hypothesis that appropriate biological responsiveness to ErbB ligands is determined by the levels of expression of specific ErbB receptor combinations within a given tissue.

11/AB/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011759356 BIOSIS NO.: 199900019016
Selective cloning of cell surface proteins involved in organ development: EGP is involved in normal epithelial differentiation
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Helmrich Angela; Barnes David; Devaux Brigitte; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p436A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

11/AB/19 (Item 19 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011756285 BIOSIS NO.: 199900015945
Distribution and function of the rat homologue of the adhesion molecule BEN during development
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qumin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Dep. Protein Chem., Genentech Inc., South San Francisco, CA 94080-4990, USA**USA
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p200A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

11/AB/20 (Item 20 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011681003 BIOSIS NO.: 199800475250
Phenylephrine, endothelin, prostaglandin F2alpha, and leukemia inhibitory factor induce different cardiac hypertrophy phenotypes in vitro
AUTHOR: King Kathleen L (Reprint); Winer Jane; Phillips David M; Quach James; Williams P Mickey; Mather Jennie P
AUTHOR ADDRESS: Genentech Inc., MS45, 1 DNA Way, South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrine 9 (1): p45-55 Aug., 1998 1998
MEDIUM: print
ISSN: 1355-008X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In these studies, we show that endothelin (ET), leukemia inhibitory factor (LIF), phenylephrine (PE), and prostaglandin F2alpha (PGF2alpha), which are all hypertrophic for neonatal rat cardiac myocytes in culture, induce distinct morphological, physiological, and genetic changes after a 48-h treatment. Transmission electron microscopy revealed differences in myofibril organization, with ET-treated cells containing the most mature-looking myofibrils and PGF2alpha- and LIF-treated cells the least. ET- and PE-treated cultures contained the same number of beating cells as control, but LIF and PGF2alpha treatment increased the number of beating cells 180%. Treatment with LIF, PE, and PGF2alpha increased the beat rate to 3.3 times that of control. After exposure to the beta-adrenergic agonist isoproterenol, the beat rate increased 50% for PGF2alpha, 54% for PE, 84% for LIF, and 125% for control. ET treatment did not increase the beat rate, nor did these cells respond to isoproterenol. ET, LIF, and PE increased the production of atrial natriuretic peptide (ANP) by threefold and PGF2alpha by 18-fold over nontreated cells. Brain natriuretic peptide (BNP) was increased fourfold by ET and PE, 16-fold by LIF, and 29-fold by PGF2alpha. Interestingly, on a pmol/L basis, only LIF induced more BNP than ANP. Treatment with all agents led to a similar pattern of gene induction: increased expression of the embryonic genes for ANP and skeletal alpha-actin, and less than a twofold change in the constitutively expressed gene myosin light chain-2, with the exception that LIF did not induce skeletal alpha-actin. Each

agent, however, induced ANP mRNA with a different time-course. We conclude that at least four distinct cardiac myocyte hypertrophy response programs can be induced in vitro. Further studies are necessary to determine whether these correlate to the different types of cardiac hypertrophy seen in vivo.

11/AB/21 (Item 21 from file: 5)

0011388375 BIOSIS NO.: 199800182622

Inhibin gene expression in a large cell calcifying Sertoli cell tumour and serum inhibin and activin levels

AUTHOR: Toppari Jorma; Kaipia Antti; Kaleva Marko; Laato Matti; De Kretser David M; Krummen Lynne A; Mather Jennie P; Salmi Toivo T
AUTHOR ADDRESS: Dep. Pediatrics and Physiol., Univ. Turku, Kiinamyllynkatu 10, FIN-20520 Turku, Finland**Finland

JOURNAL: APMIS 106 (1): p101-113 Jan., 1998 1998
MEDIUM: print
ISSN: 0903-4641
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin is a potential tumour suppressor gene product in the gonads. While inhibin gene products may have a role in tumourigenesis, serum inhibin levels can be used as a marker for ovarian tumours derived from granulosa cells. Tumours derived from Sertoli cells, testicular counterparts of granulosa cells, are rare. To assess whether inhibin could be used as a human Sertoli cell tumour marker, serum inhibin and activin levels and inhibin subunit mRNA expression in the testis were studied. Northern blot and in situ hybridization revealed abundant expression of inhibin alpha, betaA, and betaB subunit mRNAs in large cell calcifying Sertoli cell tumours found in a 12-year old boy with Carney complex. The tumours were multifocal and bilateral. Serum inhibin levels were clearly elevated at the time of the diagnosis, decreased by 50% after one of the testes was removed, and were low or undetectable after the second orchidectomy six weeks later. Activin was undetectable before the orchidectomies, while a low concentration of activin-A was measured after them. Follicle stimulating hormone (FSH) concentration increased from normal pubertal value to castration level as expected. Normal seminiferous tubules also showed inhibin subunit alpha and betaB mRNA expression, whereas inhibin betaA mRNA was expressed in normal Leydig cells. These data suggest that serum inhibin reflects Sertoli cell activity and can be used as a human tumour marker.

11/AB/22 (Item 22 from file: 5)

0011280784 BIOSIS NO.: 199800075031

Identification of an inhibin receptor in gonadal tumors from inhibin alpha-subunit knockout mice

AUTHOR: Draper Lawrence B; Matzuk Martin M; Roberts Veronica J; Cox Edward; Weiss Jeffrey; Mather Jennie P; Woodruff Teresa K (Reprint)
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JOURNAL: Journal of Biological Chemistry 273 (1): p398-403 Jan. 2, 1998

1998
MEDIUM: print
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibins and activins are dimeric proteins that are functional antagonists and are structurally related to the transforming growth factor-beta (TGFbeta) family of growth and differentiation factors. Receptors for activin and TGFbeta have been identified as dimers of serinethreonine kinase subunits that regulate cytoplasmic proteins known as Smads. Despite major advances in our understanding of activin and TGFbeta receptors and signaling pathways, little is known about inhibin receptors or the mechanism by which this molecule provides a functionally antagonistic signal to activin. Studies described in this paper indicate that an independent inhibin receptor exists. Numerous tissues were examined for inhibin-specific binding sites, including the developing embryo, in which the spinal ganglion and trigeminal ganglion-bound iodinated inhibin A. Sex cord stromal tumors, derived from male and female inhibin alpha-subunit-deficient mice, were also identified as a source of inhibin receptor. Abundant inhibin and few activin binding sites were identified in tumor tissue sections by in situ ligand binding using iodinated recombinant human inhibin A and 125I-labeled recombinant human inhibin A. Tumor cell binding was specific for each ligand (competed by excess unlabeled homologous ligand and not competed by heterologous ligand). Based on these results and the relative abundance and homogeneity of tumor tissues versus the embryonic ganglion, tumor tissues were homogenized, membrane proteins were purified, and putative inhibin receptors were isolated using an inhibin affinity column. Four proteins were eluted from the column that bind iodinated inhibin but not iodinated activin. These data suggest that inhibin-specific membrane-associated proteins (receptors) exist.

11/AB/23 (Item 23 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011227083 BIOSIS NO.: 199800021330
Characterization of cell surface proteins using antibodies raised to antigens from pancreatic cell lines
AUTHOR: Stephan Jean-Philippe; Bald Laura; Roberts Penny; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 8 (SUPPL.): p328A Nov., 1997 1997
MEDIUM: print
CONFERENCE/MEETING: 37th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 13-17, 1997; 19971213
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

11/AB/24 (Item 24 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011164585 BIOSIS NO.: 199799798645
Stimulating effect of both human recombinant inhibin A and activin A on

immature porcine Leydig cell functions in vitro
AUTHOR: Lejeune Herve; Chuzel Franck; Sanchez Pascal; Durand Philippe; Mather Jennie P; Saez Jose M (Reprint)
AUTHOR ADDRESS: INSERM-INRA U-418, Hopital Debrousse, 69322 Lyon, France** France
JOURNAL: Endocrinology 138 (11): p4783-4791 1997 1997
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In addition to the regulation of FSH secretion, it has been clearly shown that inhibin and activin have paracrine/autocrine effects in the gonads. We have studied the effect of human recombinant inhibin A and human recombinant activin A on immature porcine Leydig cells in vitro. Leydig cells were prepared by collagenase digestion of testes from 3-week-old piglets, purified on Percoll gradient, then cultured in a chemically defined medium. The cells were treated with increasing amounts of inhibin A or activin A (0.5-200 ng/ml). Direct application of either inhibin A or activin A on Leydig cells for 4 or 48 h did not stimulate basal testosterone secretion. Conversely, treatment of the cells for 48 h with either factor resulted in a dose-dependent increase in hCG-stimulated testosterone secretion (10-9 M hCG, 2 h) with a maximal effect of 2.40 +- 0.37- and 2.43 +- 0.37-fold increases for inhibin A and activin A, respectively, and these changes were associated with a slight increase in LH/hCG-binding sites (1.37 +- 0.19 and 1.24 +- 0.11-fold increases). In addition, both inhibin A and activin A enhanced messenger RNA (mRNA) levels of LH/hCG receptor (2.75 +- 0.40- and 2.53 +- 0.60-fold increases) and cytochrome P450 17-alpha-hydroxylase (6 +- 1- and 3.5 +- 0.6-fold increases), but had no effect on side-chain cleavage cytochrome P450 or cytochrome P450 aromatase mRNAs. 3-beta-Hydroxysteroid dehydrogenase mRNA levels were increased (3.1 +- 1.3-fold increase) by activin A, but not by inhibin A. However, inhibin A blocked the stimulatory action of activin A. In keeping with these changes in the steroidogenic enzyme mRNAs, both peptides enhanced the conversion of exogenous 22Rhydroxycholesterol and progesterone, but only activin A increased the conversion of dehydroepiandrosterone into testosterone. In conclusion, our findings demonstrate that both inhibin A and activin A have a stimulatory effect on immature porcine Leydig cell differentiated function in vitro. As inhibin has a stimulatory and activin has an inhibitory effect on rat Leydig cell function in vitro, the effects of these factors on Leydig cells seem to be species dependent.

11/AB/25 (Item 25 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011159174 BIOSIS NO.: 199799793234
Lindane, an inhibitor of gap junction formation, abolishes oocyte directed follicle organizing activity in vitro
AUTHOR: Li Ronghao; Mather Jennie P
AUTHOR ADDRESS: Cell Biol., Genentech Inc., 1 DNA Way, South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrinology 138 (10): p4477-4480 1997 1997
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Folliculogenesis in the ovary begins before birth with the formation of primordial follicles consisting of an oocyte in the resting meiotic prophase surrounded by a layer of undifferentiated granulosa cells. At each menstrual cycle a subset of these follicles is selected and undergoes growth and differentiation leading to ovulation and corpora lutea formation or apoptosis. We have previously described an in vitro model of rat follicular development in which the organization of three-dimensional antral-follicles requires FSH and activin A (1). Here we report that, in this system, the oocyte is required for follicle morphogenesis and requires gap junctions to direct the morphological arrangement of the surrounding granulosa cells.

11/AB/26 (Item 26 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011008654 BIOSIS NO.: 199799642714
Follicle-stimulating hormone induces terminal differentiation in a predifferentiated rat granulosa cell line (ROG)
AUTHOR: Li Ronghao; Phillips David M; Moore Alison; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Cell Biology, Genentech, 460 Point San Bruno Boulevard, South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrinology 138 (7): p2648-2657 1997 1997
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The divergent commitment of ovarian granulosa cells to either proliferation and differentiation or programmed cell death directly reflects the process of follicular dominance and atresia. This process is regulated by FSH and local paracrine factors. To further analyze the role of FSH and intraovarian factors in follicular selection, we have established a rat ovarian granulosa (ROG) cell line from prepubertal (p14) rats. ROG cells are cultured in serum-free medium with activin A, but without FSH. ROG cells bind FSH and respond to FSH by a burst of cell proliferation and increased progesterone secretion. These results support the hypothesis that activin, but not FSH, is an important factor in the maintenance of immature granulosa cells. After exposure of ROG cells to FSH, withdrawal of FSH from the cultures results in apoptotic cell death. ROG cells start active membrane blebbing by 2 h after FSH withdrawal, and most cells die within 7 h. Thus, FSH-induced ROG cells differentiate into a more mature granulosa phenotype, which is nonmitotic and dependent on FSH for survival. The ROG cell line may thus provide a good in vitro model of follicular selection.

11/AB/27 (Item 27 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011008586 BIOSIS NO.: 199799642646
Activins, inhibins, and follistatins: Further thoughts on a growing family of regulators
AUTHOR: Mather Jennie P (Reprint); Moore Alison; Li Rong-Hao
AUTHOR ADDRESS: Cell Biol., Ms 45, Genentech Inc., 460 Point San Bruno Boulevard, South San Francisco, CA 94080, USA**USA
JOURNAL: Proceedings of the Society for Experimental Biology and Medicine

215 (3): p209-222 1997 1997
ISSN: 0037-9727
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin, a feedback inhibitor of pituitary FSH secretion, and its homodimer, activin, have been the subject of a growing body of literature in the last 5 years. These factors play a role not only in endocrine feedback in the reproductive system but also in paracrine and autocrine regulation of both reproductive and nonreproductive organs, including the liver, kidney, and brain. Additionally, the messages coding for both subunits and their receptors are exquisitely regulated, both spatially and temporally, during embryonic development. The cloning of a family of activin receptors; the development of specific immunoassays for inhibins A and B, and activins A and B; the description of alpha subunit, beta subunit, and receptor loss of function transgenic mouse models; and the cloning of two new a subunit homologs have increased our understanding of the possible roles this complex family of proteins plays in development and endocrine function. This review largely confines itself to the roles of inhibins and activins in the male and female reproductive system, and is intended as an update to a 1992 review published in this journal.

11/AB/28 (Item 28 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010941828 BIOSIS NO.: 199799575888
Optimization of growth, viability, and specific productivity for expression of recombinant proteins in mammalian cells
BOOK TITLE: Methods in Molecular Biology; Recombinant gene expression protocols
AUTHOR: Mather Jennie P (Reprint); Moore Alison; Shawley Robert
BOOK AUTHOR/EDITOR: Tuan R S (Editor)
AUTHOR ADDRESS: Genetech, South San Francisco, CA, USA**USA
SERIES TITLE: Methods in Molecular Biology 62 p369-382 1997
BOOK PUBLISHER: Humana Press Inc. {a}, Suite 808, 999 Riverview Drive, Totowa, New Jersey 07512, USA
ISSN: 0097-0816 ISBN: 0-89603-333-3 (paper); 0-89603-480-1 (cloth)
DOCUMENT TYPE: Book Chapter
RECORD TYPE: Citation
LANGUAGE: English

11/AB/29 (Item 29 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010880980 BIOSIS NO.: 199799515040
Effects of temperature shift on cell cycle, apoptosis and nucleotide pools in CHO cell batch cultures
AUTHOR: Moore Alison (Reprint); Mercer Jennifer; Dutina George; Donahue Christopher J; Bauer Kenneth D; Mather Jennie P; Etcheverry Tina; Ryll Thomas (Reprint)
AUTHOR ADDRESS: Dep. Process Sci., Genentech Inc., South San Francisco, CA 94080, USA**USA
JOURNAL: Cytotechnology 23 (1-3): p47-54 1997 1997
ISSN: 0920-9069
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Temperature reduction in CHO cell batch culture may be beneficial in the production of recombinant protein and in maintenance of viability. The effects on cell cycle, apoptosis and nucleotide pools were studied in cultures initiated at 37 degree C and temperature shifted to 30 degree C after 48 hours. In control cultures maintained at 37 degree C, viable cells continued to proliferate until the termination of the culture, however, temperature reduction caused a rapid decrease in the percent of cells in S phase And accumulation of cells in G-1. This was accompanied by a concurrent reduction in U ratio (UTO/UDP-GNAc), previously shown to be a sensitive indicator of growth rate. Culture viability was extended following temperature shift, as a result of delayed onset of apoptosis, however, once initiated, the rate and manner of cell death was similar to that observed at 37 degree C. All nucleotide pools were similarly degraded at the time of apoptotic cell death. Temperature reduction to 30 degree C did not decrease the energy charge of the cells, however, the overall rate of metabolism was reduced. The latter may be sufficient to extend culture viability via a reduction in toxic metabolites and/or limitation of nutrient deprivation. However, the possibility remains that the benefits of temperature reduction in terms of both viability and productivity are more directly associated with cultures spending extended time in G-1.

11/AB/30 (Item 30 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010704715 BIOSIS NO.: 199799338775
Multiple factors control the proliferation and differentiation of rat early embryonic (Day 9) neuroepithelial cells
AUTHOR: Li Ronghao; Gao Wei-Qiang; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Genentech, 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrine 5 (2): p205-217 1996 1996
ISSN: 1355-008X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The proliferation and differentiation of neural precursor cells is largely controlled by environmental factors. By providing the factors that favor the proliferation or suppress the differentiation of this cell type, we isolated and expanded an early neuroepithelial predifferentiated cell type from E9 rat neural plate in serum-free medium. This has led to the establishment of a neural epithelial precursor (NEP) cell line. The NEP cell's properties are substantially different from those of cell lines previously derived from neural tissue at later stages of development. Initial selection and survival of this cell type requires a factor secreted by an embryonic Schwann (nrESC) cell line. Continued passage of these cells requires cell-cell contact for both survival and growth. Neural cell differentiation can be induced in this nestin positive precursor cell line by bFGF and forskolin. General neuronal markers, as well as cortical neuron-specific protein kinase C isozyme, and accumulation of glutamate and aspartate were induced in most cells. Choline acetyl-transferase was also induced in a small number of cells. When implanted into neonatal rat brain, the NEP cell line gave rise to several distinct neuronal and glial phenotypes in different regions of

the brain including cerebellar cortex and hippocampus.

11/AB/31 (Item 31 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010562216 BIOSIS NO.: 199699196276
Endogenous cardiac vasoactive factors modulate endothelin production by cardiac fibroblasts in culture
AUTHOR: King Kathleen L (Reprint); Winer Jane; Mather Jennie P
AUTHOR ADDRESS: Genentech Inc., MS50, 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrine 5 (1): p95-102 1996 1996
ISSN: 1355-008X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Endothelin, a potent vasoconstrictor, is produced by cardiac fibroblasts in culture and induces hypertrophy in cardiac myocytes. The purpose of this study was to determine whether vasoactive factors endogenous to the heart affect the production of endothelin by cultured cardiac fibroblasts. Vasoactive factors have been shown to play multiple roles in the adaptation of the heart to chronic overload, affecting both vascular tone and cell growth. Both atrial (ANP) and brain (BNP) natriuretic peptides are endogenous cardiac vasodilators and are produced by cultured myocytes in response to stimulation with endothelin. Treatment of cardiac fibroblasts with these peptides decreased endothelin production. Nitroprusside, an activator of guanylyl cyclase, decreased endothelin production indicating the involvement of cGMP in the response. Carbaprostacyclin, a stable derivative of prostacyclin, another endogenous cardiac vasodilator, also decreased endothelin production by fibroblasts. The combination of BNP and carbaprostacyclin was additive in decreasing endothelin production. In contrast; PGF2-alpha and angiotensin 11, both endogenous cardiac vasoconstrictors, increased endothelin production and overcame the inhibition induced by BNP and carbaprostacyclin. In summary, endothelin production by cardiac fibroblasts was decreased by the endogenous cardiac vasodilators ANP, BNP, and prostacyclin and increased by the endogenous vasoconstrictors PGF2-alpha and angiotensin II.

11/AB/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010562215 BIOSIS NO.: 199699196275
Cardiac fibroblasts produce leukemia inhibitory factor and endothelin, which combine to induce cardiac myocyte hypertrophy in vitro
AUTHOR: King Kathleen L (Reprint); Lai Jadine; Winer Jane; Luis Elizabeth; Yen Randy; Hooley Jeff; Williams P Mickey; Mather Jennie P
AUTHOR ADDRESS: Genentech Inc., MS45, 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrine 5 (1): p85-93 1996 1996
ISSN: 1355-008X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Cardiac fibroblasts in culture produce factor(s) that induce hypertrophy of neonatal rat ventricular myocytes in vitro. As in vivo, the myocyte hypertrophy response in culture is characterized by an increase in cell size and contractile protein content, and by the activation of embryonic genes, including the gene for atrial natriuretic peptide. The purpose of this study was to identify the factor(s) produced by fibroblasts that induce myocyte hypertrophy. The fibroblast hypertrophy activity was inhibited using a combination of the endothelin A receptor blocker BQ-123 and an antibody to leukemia inhibitory factor. The individual antagonists each caused a partial inhibition. The mRNAs for both leukemia inhibitory factor and endothelin were detected by RT-PCR analysis and the concentration of both proteins was determined to be approximately 200 pmol/L in the conditioned medium using immunoassays. Purified leukemia inhibitory factor and endothelin each induced distinctive morphological changes in the myocytes. Their combination generated a different morphology similar to that induced by fibroblast conditioned medium. Each factor also induced atrial natriuretic peptide production, but both were required for the myocytes to produce the levels measured after exposure to fibroblast conditioned medium. These results show that hypertrophy activity produced by cardiac fibroblasts in culture is a result of leukemia inhibitory factor and endothelin.

11/AB/33 (Item 33 from file: 5)

0010522215 BIOSIS NO.: 199699156275

Establishment of Schwann cell lines from normal adult and embryonic rat dorsal root ganglia

AUTHOR: Li Rong-Hao; Sliwkowski Mark X; Lo Jeannie; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Genetech Inc., 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA

JOURNAL: Journal of Neuroscience Methods 67 (1): p57-69 1996 1996
ISSN: 0165-0270
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Schwann cells, an important component of the peripheral nervous system, interact with neurons to mutually support growth and replication in the embryo and survival and differentiated function in the adult. The ability of adult Schwann cells to re-enter the cell cycle ,after nerve injury is crucial to their role in nerve repair. This ability suggests that it should be possible to obtain non-transformed, cell lines which maintain the characteristics of proliferating adult Schwann cells in vivo, as well as obtaining Schwann cells from rapidly dividing embryonic tissues. One approach to obtaining normal functionally differentiated cell lines has been to start primary cultures in serum-free medium containing growth factors and attachment proteins specifically selected to favor the replication of the cell type of interest. By culturing dispersed dorsal root ganglia on laminin, in serum-free medium with hormones and growth factors, we repeatedly generate homogenous Schwann cell cultures which yield normal Schwann cell lines from the dorsal root ganglia (DRG) of both embryonic and adult rats. These cells maintain the phenotype of Schwann cells as determined by morphology and staining for GFAP, S100, p75 NGF receptor, laminin, and MAG production in co-culture with DRG neurons.

11/AB/34 (Item 34 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010364364 BIOSIS NO.: 199698832197
Measurement of dimeric inhibin B throughout the human menstrual cycle
AUTHOR: Groome Nigel P (Reprint); Illingworth Peter J; O'Brien Martin; Pai Roger; Rodger Faye E; Mather Jennie P; McNeilly Alan S
AUTHOR ADDRESS: Sch. Biol. Molecular Sci., Oxford Brookes University, Oxford OX3 0BP, UK**UK
JOURNAL: Journal of Clinical Endocrinology and Metabolism 81 (4): p 1401-1405 1996 1996
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This report describes the development of a specific and sensitive assay for inhibin B and its application to the measurement of inhibin B concentrations in plasma during the human menstrual cycle. A monoclonal antibody raised against a synthetic peptide from the beta-B-subunit was combined with an antibody to an inhibin alpha-subunit sequence in a double antibody enzyme-linked immunosorbent assay format. The validated assay had a limit of detection of 10 pg/mL and 0.5% cross-reactivity with inhibin A. Using this immunoassay, we found that the plasma concentration of inhibin B rose rapidly in the early follicular phase to a peak of 85.2 +- 9.6 pg/mL on the day after the intercycle FSH rise, then fell progressively during the remainder of the follicular phase. Two days after the midcycle LH peak, there was a short lived peak in the inhibin B concentration (133.6 +- 31.2 pg/mL), which then fell to a low concentration (lt 20 mu-g/mL) for the remainder of the luteal phase. In contrast, the inhibin A concentration was low in the early follicular phase, rose at ovulation, and was maximal during the midluteal phase. The concentration of inhibin B in individual follicular fluid samples was 20- to 200-fold higher than the concentration of inhibin A and was highest in follicular fluid samples from the early follicular phase. Inhibin B appears to be the predominant form of inhibin in the preovulatory follicle. The different patterns of circulating inhibin B and inhibin A concentrations observed during the human menstrual cycle suggest that these forms may have different physiological roles.

11/AB/35 (Item 35 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010354710 BIOSIS NO.: 199698822543
Inhibin-B: A likely candidate for the physiologically important form of inhibin in men
AUTHOR: Illingworth Peter J (Reprint); Groome Nigel P; Byrd William; Rainey William E; McNeilly Alan S; Mather Jennie P; Bremner William J
AUTHOR ADDRESS: Med. Res. Council Reproductive Biol. Unit, Cent. Reproductive Biol., 37 Chalmers Street, Edinburgh EH3 9EW, UK**UK
JOURNAL: Journal of Clinical Endocrinology and Metabolism 81 (4): p 1321-1325 1996 1996
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin is a glycoprotein hormone that is defined on the basis of inhibition of pituitary FSH production. However, previous data have not shown any correlation between RIA measurements of inhibin and FSH in men. New enzyme-linked immunosorbent assays, specific for inhibin A, inhibin B, and inhibin pro-alpha-C-related immunoreactivity, were applied to the measurement of inhibin in 32 healthy men. Further measurements of inhibin B and pro-alpha-C-RI were carried out on groups of men exhibiting a wide range of FSH concentrations, including semen donors, infertile men, and men with elevated FSH concentrations. Inhibin A was undetectable (lt 2 pg/mL) in all men studied. The healthy men studied all had measurable concentrations of inhibin B (135.6 pg/mL; confidence interval, 108.4-169-4) and pro-alpha-C-RI (426.3 pg/mL; confidence interval, 378.4-480.2). A close negative correlation was found between the inhibin B and FSH concentrations in the semen donors (r = -0.69; P lt 0.001), the infertile men (r = -0.81; P lt 0.001), and the men with elevated FSH concentrations (r = -0.54; P lt 0.01), but not in a group of healthy volunteers (r = -0.08; P = NS). No correlation was observed between concentrations of pro-alpha-C-RI and FSH in any of the groups studied. These results strongly suggest that the physiologically important form of inhibin in men is inhibin B, which has a critical effect on FSH release. Inhibin B may offer a clinically useful serum marker of testicular function.

11/AB/36 (Item 36 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010306545 BIOSIS NO.: 199698774378
Follistatins and alpha-2-macroglobulin are soluble binding proteins for inhibin and activin
AUTHOR: Mather Jennie P
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JOURNAL: Hormone Research (Basel) 45 (3-5): p207-210 1996 1996
ISSN: 0301-0163
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin is a 32-kD dimeric glycoprotein consisting of an a subunit and one of two beta subunits (beta-A or beta-B), which was isolated and cloned on the basis of its ability to inhibit FSH release from the pituitary. Activin results from the combination of two inhibins. Activins can cause stimulation of FSH release from pituitary cells both in vitro and in vivo, and in addition are involved in embryogenesis, erythropoiesis, and reproductive function. The inhibin-related peptides, and their receptors, are present in the testis and ovary from early gestation through adulthood. An additional level of control of the activity of growth factors is afforded by specific binding which may regulate protein turnover, localization and bioactivity. To date, two distinct binding proteins for inhibin and activin have been identified, both of which are expressed in the testes and other tissues and are present in the circulation. In serum, inhibin is primarily found associated with alpha-2-macroglobulin (alpha-2M), a high-capacity, low-affinity binding protein which binds many cytokines and growth factors. Binding to alpha-2M does not appear to alter immuno- or bioactivity of inhibin or activin. The second binding protein, follistatin, is produced in many of the same tissues which produce the

activin and inhibin. This molecule may function primarily as a regulator of activin bioavailability and bioactivity. The affinity of follistatin for activin (lt 1.0 nM) is similar to that of the high-affinity activin receptors. Thus, dynamic changes in the relative levels of the amount of any of these components could act to modulate activin and inhibin bioavailability in both a developmentally and tissue-restricted pattern in the testes or ovary.

11/AB/37 (Item 37 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0010280019 BIOSIS NO.: 199698747852
Identification of Gas6 as a growth factor for human Schwann cells
AUTHOR: Li Rong-Hao; Chen Jian; Hammonds Glenn; Phillips Heidi; Armanini Mark; Wood Patrick; Bunge Richard; Godowski Paul J; Sliwkowski Mark X; Mather Jennie P (Reprint)
AUTHOR ADDRESS: MS 45, Genetech Inc., 460 Point San Bruno Boulevard, South San Francisco, CA 94080, USA**USA
JOURNAL: Journal of Neuroscience 16 (6): p2012-2019 1996 1996
ISSN: 0270-6474
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Schwann cells are one of the principal components of the peripheral nervous system. They play a crucial role in nerve regeneration and can be used clinically in the repair of injured nerves. We have established serum-free, defined culture conditions that rapidly expand adult human Schwann cells without fibroblast growth. We find that Gas6, a ligand for the Axl and Rse/Tyro3 receptor protein tyrosine kinase family, stimulates human Schwann cell growth, increasing both cell number and thymidine incorporation. Gas6 has synergistic effects with the other known human Schwann cell mitogens, heregulin/glial growth factor and forskolin. Addition of Gas6 causes phosphorylation of Axl and Rse/Tyro3 simultaneously and results in ERK-2 activation. A combination of Gas6 with heregulin and forskolin, on a defined background, supports maximal Schwann cell proliferation, while preserving the typical Schwann cell morphology and expression of the Schwann cell markers S-100, glial fibrillary acidic protein, and low-affinity nerve growth factor receptor. Gas6 mRNA is present in both spinal motor neurons and large neurons of the dorsal root ganglia, and neural injury has been reported to upregulate Rse/Axl in the Schwann cell. This is the first demonstration of a potentially important biological role for the human Gas6/RseAxl system.

11/AB/38 (Item 38 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009997116 BIOSIS NO.: 199598464949
Apoptosis in CHO cell batch cultures: Examination by flow cytometry
AUTHOR: Moore Alison (Reprint); Donahue Christopher J; Hooley Jeff; Stocks Diana L; Bauer Kenneth D; Mather Jennie P
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JOURNAL: Cytotechnology 17 (1): p1-11 1995 1995
ISSN: 0920-9069

DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Chinese hamster ovary cells grown under conditions which are optimal for the production of a genetically engineered protein in batch culture, lose significant viability shortly after entering the stationary phase. This cell death was investigated morphologically and was found to be almost exclusively via apoptosis. Furthermore, cells were analyzed by flow cytometry using a fluorescent DNA end-labeling assay to label apoptotic cells, in conjunction with cell cycle analysis using propidium iodide. Apoptotic cells could be detected by this method, and by the radioactive end-labeling of extracted DNA, on all days of culture from day 1 to day 7; however, the degree of apoptotic cell death increased dramatically when the cells entered the stationary phase, rising to 50-60% of the total cell number at the termination of the culture. Flow cytometric analysis showed that the majority of cells underwent apoptosis whilst in G-1/G-0 and formed an apoptotic population with high DNA FITC end-labeling and hypodiploid propidium iodide binding. Additionally, the ability or inability to secrete specific protein products did not appear to interfere with the development of the apoptotic population with time.

11/AB/39 (Item 39 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009777441 BIOSIS NO.: 199598245274
Inhibin, activin and the female reproductive axis
BOOK TITLE: Annual Review of Physiology
AUTHOR: Woodruff Teresa K; Mather Jennie P
BOOK AUTHOR/EDITOR: Hoffman J F (Editor)
AUTHOR ADDRESS: Discovery Res., Genentech Inc., South San Francisco, CA 94080, USA**USA
SERIES TITLE: Annual Review of Physiology 57 p219-244 1995
BOOK PUBLISHER: Annual Reviews Inc. {a}, P.O. Box 10139, 4139 El Camino Way, Palo Alto, California 94306, USA
ISSN: 0066-4278 ISBN: 0-8243-0357-1
DOCUMENT TYPE: Book; Book Chapter; Literature Review
RECORD TYPE: Citation
LANGUAGE: English

11/AB/40 (Item 40 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009727619 BIOSIS NO.: 199598195452
Activin promotes ovarian follicle development in vitro
AUTHOR: Li Ronghao (Reprint); Phillips David M; Mather Jennie P
AUTHOR ADDRESS: Genentech, Inc., MS 45, 460 Point San Bruno Boulevard, South San Francisco, CA 94080, USA**USA
JOURNAL: Endocrinology 136 (3): p849-856 1995 1995
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Activin is a protein originally isolated from follicular fluid as a factor stimulating FSH release from the pituitary. The present

experiments support the hypothesis that activins may also regulate follicle development by autocrine/paracrine mechanisms. Granulosa-oocyte complexes were isolated by collagenase/dispase dispersion of ovaries from 14- or 21-day-old rats and cultured in serum-free medium. Within 24 h, the cells had spread to form a monolayer. Hormones and growth factors were added at this time. Cell number and thymidine incorporation were measured after an additional 72 h. In the presence of insulin and transferrin, activin-A increased both granulosa cell number and thymidine incorporation more than 2-fold. This effect could be inhibited by follistatin, an activin-binding protein. In addition, activin-A, in the presence of FSH, induced reorganization of follicular structures from monolayer culture of cells from 14-day-old rats and caused cells from primary follicles to develop into large follicle-like structures. These structures contained oocytes, a cumulus layer, an antrum, and a multilayered follicular wall with a diameter of more than 1 mm. Electron microscopy revealed that the cells in the follicle-like structure were connected by gap junctions. Oocytes showed a mature morphology and had closely associated cumulus layers. Dissociation of the follicular wall in these follicle-like structures was induced by the addition of LH, resembling the induction of ovulation in vivo. The findings are important for understanding follicular development and atresia.

11/AB/41 (Item 41 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009460557 BIOSIS NO.: 199497481842
The effect of exogenous recombinant human activin A on pituitary and ovarian hormone secretion and ovarian folliculogenesis in female rats and monkeys
BOOK TITLE: International Congress Series; Ovulation induction: Basic science and clinical advances
AUTHOR: Woodruff Teresa K (Reprint); Molskness Theodore A; Dahl Kristine D; Mather Jennie P; Stouffer Richard L
BOOK AUTHOR/EDITOR: Filicori M (Editor); Flamigni C (Editor)
AUTHOR ADDRESS: Genentech Inc., 460 Pt. San Bruno Blvd., South San Francisco, CA 94080, USA**USA
SERIES TITLE: International Congress Series 1046 p57-63 1994
BOOK PUBLISHER: Excerpta Medica, 305 Keizersgracht, PO Box 1126, Amsterdam, Netherlands
Excerpta Medica, New York, New York, USA
CONFERENCE/MEETING: Meeting Palm Beach, Florida, USA January 20-22, 1994; 19940120
ISSN: 0531-5131 ISBN: 0-444-81696-8
DOCUMENT TYPE: Book; Meeting; Book Chapter; Meeting Paper
RECORD TYPE: Citation
LANGUAGE: English

11/AB/42 (Item 42 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009372966 BIOSIS NO.: 199497394251
Effects of insulin and activin A on Sertoli cell- germ cell co-cultures
AUTHOR: Moore Alison; Mather Jennie P
AUTHOR ADDRESS: Genetech, Inc., South San Francisco, CA 94080, USA**USA
JOURNAL: Biology of Reproduction 50 (SUPPL. 1): p79 1994 1994
CONFERENCE/MEETING: Twenty-seventh Annual Meeting of the Society for the

Study of Reproduction Ann Arbor, Michigan, USA July 24-27, 1994; 19940724
ISSN: 0006-3363
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

11/AB/43 (Item 43 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009260953 BIOSIS NO.: 199497282238
Inhibins, activins, their binding proteins and receptors: Interactions underlying paracrine activity in the testis
AUTHOR: Moore Alison; Krummen Lynne A; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Genetech Inc., 460 Point San Bruno Blvd., South San Francisco, CA 94080, USA**USA
JOURNAL: Molecular and Cellular Endocrinology 100 (1-2): p81-86 1994 1994
ISSN: 0303-7207
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The inhibin-related peptides are present in the testis from early gestation through adulthood. They are produced from multiple testicular sites in a highly regulated manner, suggesting important paracrine roles. Similarly, receptors for these peptides are located in specific stages of the seminiferous tubule and on particular cell types, and an additional level of control is afforded by specific binding proteins, such as follistatin, which may regulate bioavailability. The actions of these factors include the modulation of interstitial cell function and the increase of spermatogonial proliferation in vitro. It thus appears that activin and inhibin are significant factors in the local control of testicular function.

11/AB/44 (Item 44 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009209918 BIOSIS NO.: 199497231203
Localization of inhibin and activin binding sites in the testis during development by in situ ligand binding
AUTHOR: Krummen Lynne A (Reprint); Moore Alison; Woodruff Teresa K; Covello Robin; Taylor Robin; Working Peter; Mather Jennie P
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JOURNAL: Biology of Reproduction 50 (4): p734-744 1994 1994
ISSN: 0006-3363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin and activin are related proteins thought to be potential paracrine regulators of testicular development and maintenance of spermatogenesis. Messenger RNA and proteins immunologically related to both factors have been identified in the adult testis. However, the role(s) of these factors in paracrine regulation of testicular function is poorly understood. To identify potential targets for inhibin and activin in immature and adult testis, we used in situ binding of

(125I)-labeled ligands to localize and describe the distribution of binding sites for inhibin and activin in testes of 15-, 18-, 21-, 30-, 45-, and 60-day-old rats. Nonspecific binding was defined as that occurring in the presence of a 1000-fold excess of unlabeled recombinant human (rh) inhibin or activin. (125I)-Inhibin was found to bind to interstitial cells throughout development. Inhibin binding was shown to co-localize with cells that showed positive staining for 3-beta-hydroxysteroid dehydrogenase (3-beta-HSD). Competition studies demonstrated that this binding was indeed specific for inhibin. In contrast, (125I)-activin showed two distinct patterns of binding. First, (125I)-activin was shown to bind in a non-stage-dependent manner to cells located in the basal compartment of the seminiferous tubules in testis obtained from animals of ail ages studied. Binding of (125I)-activin in the periphery of the tubule could be inhibited entirely by coincubation with excess unlabeled activin and partially with excess unlabeled inhibin. The ability of inhibin to compete with activin for binding appeared to be more pronounced in younger animals. In 45- and 60-day-old animals, a second stage-dependent component of (125I)-activin binding was also apparent. This binding was localized to spermatids found in stage VII-VIII tubules and was inhibited by the presence of excess activin, but not inhibin. These results indicate that inhibin can bind specifically to testicular interstitial cells throughout development and may be an important regulator of Leydig cell testosterone production or interstitial cell function. In contrast, activin appears to bind in a specific and stage-dependent manner to receptors or high-affinity binding proteins on spermatids as well as to sites on the periphery of all seminiferous tubules. These results support the hypothesis that both activin and inhibin may act at several levels to regulate proliferation or differentiation of germ and Sertoli cell function as well as to modulate interstitial cell activity.

11/AB/45 (Item 45 from file: 5)

0009209904 BIOSIS NO.: 199497231189

Systemic and intraluteal infusion of inhibin A or activin A in rhesus monkeys during the luteal phase of the menstrual cycle

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JOURNAL: Biology of Reproduction 50 (4): p888-895 1994 1994
ISSN: 0006-3363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The endocrine or local actions of inhibin-related peptides synthesized by the primate corpus luteum (CL) remain undefined. This in vivo study was designed to determine whether exogenous inhibin or activin modulates pituitary gonadotropin secretion and the functional life span of the CL during the luteal phase of the menstrual cycle. Beginning at midluteal phase of the cycle, either vehicle or 1 mu-g/h of recombinant human inhibin A or activin A (n = 3-6 per treatment group) was infused into rhesus monkeys via the jugular vein (i.e., peripheral infusion) or directly into the CL (i.e., intraluteal infusion) by means of an osmotic minipump for 7-14 days. Daily samples of saphenous venous serum were assayed for estradiol (E) and progesterone (P) content by RIA, and for

FSH and LH levels by bioassay. Intraluteal infusion of inhibin or activin did not alter circulating P levels or the length of the luteal phase compared to those values in vehicle-infused controls. likewise, LH levels were not different between the three groups. However, FSH levels declined progressively during inhibin infusion to 26% of pretreatment levels (p lt 0.05), whereas FSH levels in vehicle-infused controls were unchanged for several days and then rose (p lt 0.05) to peak levels around menses. FSH levels did not change significantly during activin infusion into the CL Although similar results were obtained in monkeys receiving peripheral or intraluteal infusions of inhibin, events following the peripheral infusion of activin were markedly different from those during intraluteal administration. Peripheral activin treatment caused a sustained reduction in serum LH and P levels (p lt 0.05) and shortened the length of the luteal phase compared to that in control cycles (9.2 +- 0.3 vs. 14.7 +- 1.3 days, p lt 0.05). The data provide the first evidence that exogenous inhibin selectively suppresses FSH levels in primates during the mid-to-late luteal phase of the menstrual cycle without altering the functional life span of the CL In contrast, exogenous activin infused systemically caused rapid, premature regression of the CL Since intraluteal infusion did not alter luteal function, peripheral actions, including suppression of LH (but not FSH) levels, may be implicated in the luteolytic effect of activin.

11/AB/46 (Item 46 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009051343 BIOSIS NO.: 199497072628
In situ ligand binding of recombinant human (125I) activin-A and recombinant human (1-2-5I)inhibin-A to the adult rat ovary
AUTHOR: Woodruff Teresa K (Reprint); Krummen Lynne; McCray Glynis; Mather Jennie P
AUTHOR ADDRESS: Dep. Cell Culture Research Development, Genentech Inc., South San Francisco, CA 940809, USA**USA
JOURNAL: Endocrinology 133 (6): p2998-3006 1993 1993
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibin and activin are hormones produced by ovarian follicles. Specific ovarian cells that bind iodinated recombinant human (rh)activin-A and rh-inhibin-A were identified by in situ ligand binding. Iodinated rh-molecules were incubated with tissue sections from ovaries, uterus, and oviduct, collected on the mornings of metestrus, diestrus, proestrus, and estrus and the evening of proestrus. Additionally, inhibin/activin subunit mRNA and follistatin mRNA accumulation was examined by in situ hybridization of radiolabeled antisense riboprobes. The cellular site of activin/inhibin binding could thus be colocalized to the cellular site of ligand mRNA and binding protein mRNA production. The association of both (125I)rh-activin-A and (125I) rh-inhibin-A with specific cell types varied across the rat estrous cycle. Nonspecific binding was evaluated by competition with a 1000-fold excess of homologous ligand, and low affinity association of the heterologous ligand was evaluated by competition with a 1000-fold excess of heterologous ligand. (125I)rh-activin-A binding was more widespread than was (125I)rh-inhibin-A binding under our experimental conditions. (125I)rh-Activin-A bound to the granulosa cells of all stages of follicles: unrecruited, growing, and Graafian follicles. Thecal cell

binding was found in developing follicles (350-500 mu-m). The granulosa cells of stimulated follicles (evening of proesterus) bound less (125I) rh-activin-A than those of unstimulated follicles. (125I)rh-Activin-A binding was also associated with antral fluid of follicles in each size class. Although early atretic follicles retained some (125I)rh-activin-A binding, late atretic follicles did not bind (125I)rh-activin-A. Corpus luteum present on metestrus and diestrus bound (11-25I)rh-activin-A; however, corpus lutea present on proestrus and estrus bound little or no (11-25I)rh-activin-A. (125I)rhActivin-A-binding sites were also present in the uterus and oviduct in a cycle-dependent manner. The highest levels of binding were found in the muscle wall of the uterus and the epithelial lining of the thick-walled portion of the uterus on metestrus and diestrus. In addition, (125I)rh-activin-A bound to the cumulus-ooctye complex present in the oviduct on metestrus, but did not bind to the oocytes present in developing follicles. Binding of (125I)rh-inhibin-A was restricted to the antral granulosa cells of 450- to 500-mu-m follicles. No other ovarian, uterine, or oviduct cells bound (125I)rh-inhibin-A. (125I)rh-Activin-A and (125I)rh-inhibin-A ligand binding was associated primarily with follicles coexpressing inhibin/activin subunit and follistatin mRNA. The current data support the hypothesis that inhibin/activin have paracrine activities in the ovaries.

11/AB/47 (Item 47 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008897737 BIOSIS NO.: 199396062153
Human recombinant activin-A alters pituitary luteinizing hormone and follicle-stimulating hormone secretion, follicular development, and steroidogenesis, during the menstrual cycle in rhesus monkeys
AUTHOR: Stouffer Richard L (Reprint); Woodruff Teresa K; Dahl Kristine D; Hess David L; Mather Jennie P; Molskness Theodore A
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JOURNAL: Journal of Clinical Endocrinology and Metabolism 77 (1): p241-248 1993
ISSN: 0021-972X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Activin, a stimulator of pituitary FSH secretion in nonprimate species, may also act in the ovary to modulate follicular development. To examine whether activin has similar actions in primates, female rhesus monkeys (n = 3/treatment) exhibiting regular menstrual cycles received sc injections of either vehicle or 60 mu-g/kg recombinant human activin-A at 0800 and 1600 h for 1 (acute) or 7 (chronic) days beginning in the early follicular phase. The vehicle-treated monkeys displayed menstrual cycles of normal length, with the follicular (11.3 +- 1.3 days, mean +- SE) and luteal (16.6 +- 1.8 days) phases demarcated by midcycle peaks in serum estradiol (E) and bioactive LH. After the first activin injection, levels of human activin A peaked at 90 ng/mL within 1 h and returned to baseline before the second injection 8 h later. Although serum E and FSH levels did not change, LH increased (273%, P lt 0.05) within 8 h. Acute activin treatment increased (P lt 0.05) serum E within 24 h to levels (1290 +- 330 pmol/L) typically observed at midcycle. With chronic treatment, serum E peaked on day 2 (2580 +- 338 pmol/L; P lt 0.05), then declined and rose to a second peak (1680 +- 279 pmol/L) on day 5. During chronic activin

treatment, LH levels peaked on day 2 (603 +- 270 ng/mL; P lt 0.05 compared to day 0, 15 +- 7 ng/mL) whereas FSH increased progressively until day 5 (937 +- 320 ng/mL; P lt 0.05 compared to day 0, 169 +- 59 ng/mL). After acute or chronic activin, the expected midcycle rises in serum E and gonadotropins were delayed to greater than or equal to day 20 (n = 4) or did not occur before menses (n = 2). Although an enlarged ovary with one greater than or equal to 4-mm follicle was observed by laparoscopy during the late follicular phase in vehicle-treated monkeys, medium-to-large follicles were not visible on ovaries during chronic activin treatment or later at the expected midcycle interval in activin-treated monkeys. Similar hormonal and ovarian events were obtained after activin treatment of amenorrheic monkeys having serum FSH, LH, and E levels that were comparable to those at menses in spontaneous menstrual cycles. Thus, exogenous activin stimulates pituitary LH and FSH secretion and ovarian estrogen secretion during the early follicular phase in intact monkeys. However, acute or chronic activin treatment did not promote complete follicular development and disrupted subsequent events in the menstrual cycle. The study identifies for the first time potent actions and possible roles for activin in the normal and dysfunctional reproductive cycle in primates.

11/AB/48 (Item 48 from file: 5)

0008881321 BIOSIS NO.: 199396045737

Follistatin modulates activin activity in a cell- and tissue-specific manner

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JOURNAL: Endocrinology 132 (6): p2732-2734 1993
ISSN: 0013-7227
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RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The high affinity activin-binding protein, follistatin, has recently been shown to block activin-stimulated activities in several in vitro systems. In the present study we sought to extend these observations and investigate the effects of follistatin on the activity of activin in stimulating the re-aggregation of Sertoli cell monolayers and proliferation of testicular germ cells, as measured by incorporation of (3H)-thymidine in vitro. Germ-Sertoli cell cocultures prepared from 21 day old rats were treated with media alone or media containing recombinant human (rh) activin A or rh activin B with or without follistatin, the low affinity activin-binding protein, alpha-2 macroglobulin, or a monoclonal antibody (mAB) known to block activin B activity. Follistatin blocked the ability of activin A to stimulate reaggregation of Sertoli cell monolayers when present at a 2-fold ratio (wt/wt) to activin. However, in these same cultures, follistatin bad no effect on the ability of activin A to stimulate (3H)-thymidine incorporation. In activin B-treated cultures, both responses could be blocked by the addition of a neutralizing mAB directed against activin B. These results suggest that follistatin can modulate activin action in a cell-type specific fashion, and that this protein may play an important role in regulating the bioavailability of activin.

11/AB/49 (Item 49 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008862496 BIOSIS NO.: 199396026912
Development of a specific and sensitive two-site enzyme linked immunosorbent assay for measurement of inhibin A in serum
AUTHOR: Baly Deborah L (Reprint); Allison David E; Krummen Lynne A; Woodruff Teresa K; Soules Michael R; Chen Sharon A; Fendly Brian M; Bald Laura N; Mather Jennie P; Lucas Catherine
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JOURNAL: Endocrinology 132 (5): p2099-2108 1993
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A polyclonal chicken antiserum against purified 32-kilodalton (kDa) recombinant inhibin-A (rh-InhA) and two monoclonal antibodies (mAb) against either rh-InhA (11B5) or 28-kDa recombinant activin-A (rh-ActA; 9A9) were used to develop three sensitive InhA enzyme-linked immunosorbent assays (ELISAs). The sensitivity of an ELISA using affinity-purified chicken anti-rh-InhA (Ck) for both coat and capture (Ck/Ck) averaged 78 +- 3 pg/ml, while the mAb/Ck ELISAs (11B5/Ck or 9A9/Ck) averaged 100 +- 6 pg/ml in a 10% serum matrix, with intra- and interassay coefficients of variation of 2-5% and 8-10%, respectively, for all assays. The ELISA formats did not cross-react with purified rh-ActA or recombinant human transforming growth factor-b-1 or detect any immunoreactive proteins in medium conditioned by cell lines expressing rh-ActA or recombinant human transforming growth factor-beta-1. The Ck/Ck ELISA detected significant amounts of immunoreactivity in medium from cells expressing the free alpha-subunit of inhibin and recombinant inhibin-B (rh-InhB). In contrast, the mAb/Ck ELISAs showed no cross-reactivity to medium conditioned by these two cell lines. All three ELISA formats detected rh-InhA added to either human or rat serum in vitro or serum from rats injected with rh-InhA. The Ck/Ck and 9A9/Ck ELISAs successfully quantitated inhibin in sera from patients undergoing ovulation induction and in rats (with or without sc administration of pregnant female serum gonadotropin). The 11B5/Ck ELISA appeared to be specific for the 32-kDa form of inhibin, while the 9A9/Ck ELISA was useful in quantitating inhibin-A in biological fluids, with little cross-reactivity to free alpha-chain or inhibin-B.

11/AB/50 (Item 50 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008770085 BIOSIS NO.: 199395072351
Identification and characterization of binding proteins for inhibin and activin in human serum and follicular fluids
AUTHOR: Krummen Lynne A (Reprint); Woodruff Teresa K; Deguzman Geralyn; Cox Edward T; Baly Deborah L; Mann Elizabeth; Garg Shaily; Wong Wai-Lee; Cossum Paul; Mather Jennie P
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JOURNAL: Endocrinology 132 (1): p431-443 1993
ISSN: 0013-7227
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Inhibins and activins are produced by a variety of tissues and may have important endocrine and paracrine roles in development, reproduction, and hematopoeisis. However, little is known regarding the physical properties or concentrations of inhibin and activin in biological fluids. Binding proteins for inhibin or activin in serum or at production or target sites may have important implications for restricting the bioactivity of these hormones and may alter the immunoreactivity of these molecules in biological fluids. The objective of this study was to identify inhibin- and activin-binding proteins in human serum (HS) and follicular fluid (hFF) and determine the ability of these proteins to alter biological or immunological activity. In HS, (125I)activin and inhibin bound to a protein identified as alpha-2-macroglobulin (alpha-2M) using three criteria: 1) (125I)inhibin and activin bind purified alpha-2M, but not several other serum proteins tested; 2) complexes formed by (125I)inhibin and activin in HS and in the presence of purified alpha-2M elute with similar retention times on HPLC; and 3) preadsorption of HS with alpha-2M antiserum inhibits inhibin and activin binding to this protein while antiserum directed against follistatin or other serum proteins had no effect. A small amount of a lower mol wt (125I)activin-follistatin complex was also found in HS. This complex eluted with a retention time similar to that of activin bound to purified porcine follistatin. Binding of inhibin to follistatin could not be detected in HS. In contrast, follistatin was the major binding protein of both activin and inhibin in hFF. Concentrations up to 100 mu-g/ml purified alpha-2M had no effect on the bioactivity or immunoreactivity of either inhibin or activin. In contrast, follistatin inhibited both activin-stimulated pituitary FSH release and K562 hemoglobin production as well as antiserum binding in a specific activin-A immunoassay. Follistatin did not interfere with inhibin immunodetection. These data indicate that two inhibin- and activin-binding proteins are present in different relative amounts of HS and hFF. alpha-2M, the primary binding protein in HS, did not alter inhibin or activin bio- or immunoreactivity under the conditions of these experiments, while follistatin, the major binding protein in hFF, may mask activin's bio- and immunoactivities.

11/AB/51 (Item 51 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0008758496 BIOSIS NO.: 199395060762
Comparison of functional response of rat, macaque, and human ovarian cells in hormonally defined medium
AUTHOR: Woodruff Teresa K (Reprint); Battaglia Jane; Bowdidge Anne; Molskness Ted A; Stouffer Richard L; Cataldo Nicholas A; Giudice Linda C; Orly Joseph; Mather Jennie P
AUTHOR ADDRESS: 460 Point San Bruno Blvd., South San Francisco, Calif. 94080, USA**USA
JOURNAL: Biology of Reproduction 48 (1): p68-76 1993
ISSN: 0006-3363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A serum-free medium has been developed which supports in vitro function by ovarian cells derived from rat, monkey, and human tissue. This granulosa cell medium (GCM) consists of Dulbecco's Modified Eagle's

Medium: Ham's F-12 medium (1:1, v:v) supplemented with insulin, transferrin, aprotinin, selenium, fibronectin, penicillin, and streptomycin. Ovarian cells from three species were compared: rat, macaque, and human. Four types of ovarian cultures were examined: 1) purified granulosa cell cultures and 2) co-cultures containing granulosa-theca-stroma cells, 3) luteal cells, and 4) granulosa-lutein (harvested from in vitro fertilization cultures) cells. Each cell type was characterized by its response to FSH or hCG when cultured in GCM. Morphologic responses to FSH were observed in GCM in rat granulosa and granulosa-theca-stroma cell cultures, macaque and human granulosa-lutein cells, and human granulosa-theca-stroma cell cultures. The FSH-stimulated cells retracted and became rounded, leaving long intercellular connections. Luteal cells did not retract in response to FSH, and the cells remained firmly attached to the fibronectin matrix. Steroidogenic regulation of the GCM-cultured ovarian cells was monitored following stimulation of the cultures with FSH. The ability of the cells to aromatize testosterone was first examined. Rat granulosa cell cultures and granulosa-theca-stroma cell cultures, macaque granulosa-lutein cell cultures, and human granulosa-theca-stroma cell cultures all accumulated estradiol when given FSH and testosterone for 48 h. Moreover, these cell types as well as human luteal cells were able to metabolize 25-hydroxy (1, 2-3H)cholesterol to various steroid metabolites. The data indicate that GCM supports normal granulosa cell morphologic response to FSH. Moreover, the aromatase and side-chain cleavage enzymes remain active and under gonadotropin regulation when the cells maintained in GCM. Lastly, cells isolated from three species (rat, macaque, and human) respond similarly to FSH when maintained in GCM.

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TYPE S19/MEDIUM ,AB/1-19
>>>No matching display code(s) found in file(s): 359

19/AB/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014947717 BIOSIS NO.: 200400318474
Fibroblast growth factor 19 increases metabolic rate I and reverses dietary and leptlin-deficient diabetes
AUTHOR: Fu Ling; John Linu M; Adams Sean H; Yu Xing Xian; Tomlinson Elizabeth; Renz Mark; Williams P Mickey; Soriano Robert; Corpuz Racquel; Moffat Barbara; Vandlen Richard; Simmons Laura; Foster Jessica; Stephan Jean-Philippe; Tsai Siao Ping; Stewart Timothy A (Reprint)
AUTHOR ADDRESS: Dept Biol Mol, Genentech Inc, 1 DNA Way, San Francisco, CA, 94080, USA**USA
AUTHOR E-MAIL ADDRESS: timstewart@yahoo.com
JOURNAL: Endocrinology 145 (6): p2594-2603 June 2004 2004
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Hormonal control of metabolic rate can be important in regulating the imbalance between energy intake and expenditure that underlies the development of obesity. In mice fed a high-fat diet, human fibroblast growth factor 19 (FGF19) increased metabolic rate (1.53 +/- 0.06 liters O2/hcntdotkg0.75 (vehicle) vs. 1.93 +/- 0.05 liters O2/hcntdotkg0.75 (FGF19); P < 0.001) and decreased respiratory quotient (0.82 +/- 0.01 (vehicle) vs. 0.80 +/- 0.01 (FGF19); P < 0.05). In contrast to the vehicle-treated mice that gained weight (0.14 +/- 0.05 g/mouse.d), FGF19-treated mice lost weight (-0.13 +/- 0.03 g/mouse-d; P < 0.001) without a significant change in food intake. Furthermore, in addition to a reduction in weight gain, treatment with FGF19 prevented or reversed the diabetes that develops in mice made obese by genetic ablation of brown adipose tissue or genetic absence of leptin. To explore the mechanisms underlying the FGF19-mediated increase in metabolic rate, we profiled the FGF19-induced gene expression changes in the liver and brown fat. In brown adipose tissue, chronic exposure to FGF19 led to a gene expression profile that is consistent with activation of this tissue. We also found that FGF19 acutely increased liver expression of the leptin receptor (1.8-fold; P < 0.05) and decreased the expression of acetyl coenzyme A carboxylase 2 (0.6-fold; P < 0.05). The gene expression changes were consistent with the experimentally determined increase in fat oxidation and decrease in liver triglycerides. Thus, FGF19 is able to increase metabolic rate concurrently with an increase in fatty acid oxidation.

19/AB/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014819690 BIOSIS NO.: 200400187376
Albumin stimulates the accumulation of extracellular matrix in renal tubular epithelial cells.
AUTHOR: Stephan Jean-Philippe (Reprint); Mao Weiguang; Filvaroff Ellen; Cai Liping; Rabkin Ralph; Pan GuoHua
AUTHOR ADDRESS: Assay and Automation Technology Department, Genentech,

Inc., 1 DNA Way, M/S-98, South San Francisco, CA, 94080-4990, USA**USA
AUTHOR E-MAIL ADDRESS: stephanj@gene.com
JOURNAL: American Journal of Nephrology 24 (1): p14-19 January-February 2004 2004
MEDIUM: print
ISSN: 0250-8095
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The accumulation of a large amount of plasma proteins in the urine, previously regarded as a marker of glomerular damage, is now recognized as a mediator of tubulointerstitial damage. Using an in vitro approach, several key extracellular matrix (ECM) proteins were analyzed after treatment of primary human renal proximal tubular epithelial cells with fatty acid free human albumin. We demonstrate that human albumin stimulates the accumulation of ECM proteins by proximal tubular epithelial cells through a post-transcriptional mechanism. Albumin induced a significant increase in tissue inhibitor of metalloproteinases (TIMP)-1 and TIMP-2. Taken together, our data suggest that ECM protein accumulation in response to albumin resulted partly from inhibition of ECM degradation. Addition of transforming growth factor beta (TGF-beta)-specific neutralizing antibody failed to alter ECM protein levels after albumin treatment, indicating that the albumin-induced increase in ECM is TGF-beta independent. In conclusion, we have shown that exposure of cultured human proximal tubular cell to albumin leads to the TGF-beta-independent accumulation of ECM proteins, suggesting that albumin may be a contributing factor to the progression of kidney fibrosis in proteinuric states.

19/AB/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013913902 BIOSIS NO.: 200200507413
Development of a frozen cell array as a high-throughput approach for cell-based analysis
AUTHOR: Stephan Jean Philippe (Reprint); Schanz Silvia; Wong Anne; Schow Peter; Wong Wai Lee T
AUTHOR ADDRESS: Department of Assay and Automation Technology, Genentech, Inc., 1 DNA Way, South San Francisco, CA, 94080, USA**USA
JOURNAL: American Journal of Pathology 161 (3): p787-797 September, 2002 2002
MEDIUM: print
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Recent advances in molecular biology, human genetics, and functional genomics tremendously increase the number of molecular targets available for potential therapeutic and diagnostic use. To complement DNA array data, cost-efficient high-throughput technologies providing reliable information at the protein level need to be developed. Here we describe the generation of a frozen cell array that required the use of single cell suspensions and could serve various applications such as the analysis of specific antibody or ligand binding to a large panel of different cell types. As an example, binding of an anti-human epithelial cell adhesion molecule monoclonal antibody to 24 different cell lines has

been analyzed using the cell array and compared to the data generated by fluorescence-activated cell sorting. The reliability and flexibility of our frozen cell array technology is compatible with the needs of high-throughput screening for drug discovery and target validation.

19/AB/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013759299 BIOSIS NO.: 200200352810
A mouse model of hepatocellular carcinoma: Ectopic expression of fibroblast growth factor 19 in skeletal muscle of transgenic mice
AUTHOR: Nicholes Katrina; Guillet Susan; Tomlinson Elizabeth; Hillan Kenneth; Wright Barbara; Frantz Gretchen D; Pham Thinh A; Dillard-Telm Lisa; Tsai Siao Ping; Stephan Jean-Philippe; Stinson Jeremy; Stewart Timothy; French Dorothy M (Reprint)
AUTHOR ADDRESS: Genentech, Inc., 1 DNA Way, MS 72B, South San Francisco, CA, 94080, USA**USA
JOURNAL: American Journal of Pathology 160 (6): p2295-2307 June, 2002 2002
MEDIUM: print
ISSN: 0002-9440
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Most mouse models of hepatocellular carcinoma have expressed growth factors and oncogenes under the control of a liver-specific promoter. In contrast, we describe here the formation of liver tumors in transgenic mice overexpressing human fibroblast growth factor 19 (FGF19) in skeletal muscle. FGF19 transgenic mice had elevated hepatic alpha-fetoprotein mRNA as early as 2 months of age, and hepatocellular carcinomas were evident by 10 months of age. Increased proliferation of pericentral hepatocytes was demonstrated by 5-bromo-2'-deoxyuridine incorporation in the FGF19 transgenic mice before tumor formation and in nontransgenic mice injected with recombinant FGF19 protein. Areas of small cell dysplasia were initially evident pericentrally, and dysplastic/neoplastic foci throughout the hepatic lobule were glutamine synthetase-positive, suggestive of a pericentral origin. Consistent with chronic activation of the Wingless/Wnt pathway, 44% of the hepatocellular tumors from FGF19 transgenic mice had nuclear staining for beta-catenin. Sequencing of the tumor DNA encoding beta-catenin revealed point mutations that resulted in amino acid substitutions. These findings suggest a previously unknown role for FGF19 in hepatocellular carcinomas.

19/AB/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013727256 BIOSIS NO.: 200200320767
Albumin stimulates the accumulation of extracellular matrix and latent TGF-Beta in primary human proximal tubular epithelial cells
AUTHOR: Mao Weiguang (Reprint); Stephan Jean Philippe; Filvaroff Ellen (Reprint); Cai Lingping (Reprint); Rabkin Ralph; Pan GuoHua (Reprint)
AUTHOR ADDRESS: Endocrinology, Genentech, Inc., South San Francisco, CA, USA**USA
JOURNAL: Journal of the American Society of Nephrology 12 (Program and Abstract Issue): p710A September, 2001 2001
MEDIUM: print

CONFERENCE/MEETING: ASN (American Society of Nephrology)/ISN (International Society of Nephrology) World Congress of Nephrology San Francisco, CA, USA October 10-17, 2001; 20011010
ISSN: 1046-6673
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

19/AB/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013680298 BIOSIS NO.: 200200273809
Transgenic mice expressing human fibroblast growth factor-19 display increased metabolic rate and decreased adiposity
AUTHOR: Tomlinson Elizabeth; Fu Ling; John Linu; Hultgren Bruce; Huang Xiaojian; Renz Mark; Stephan Jean Philippe; Tsai Saio Ping; Powell-Braxton Lyn; French Dorothy; Stewart Timothy A (Reprint)
AUTHOR ADDRESS: Genentech, Inc., 1 DNA Way, South San Francisco, CA, 94080, USA**USA
JOURNAL: Endocrinology 143 (5): p1741-1747 May, 2002 2002
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The fibroblast growth factors (FGFs), and the corresponding receptors, are implicated in more than just the regulation of epithelial cell proliferation and differentiation. Specifically, FGF23 is a regulator of serum inorganic phosphate levels, and mice deficient in FGF receptor-4 have altered cholesterol metabolism. The recently described FGF19 is unusual in that it is nonmitogenic and appears to interact only with FGF receptor-4. Here, we report that FGF19 transgenic mice had a significant and specific reduction in fat mass that resulted from an increase in energy expenditure. Further, the FGF19 transgenic mice did not become obese or diabetic on a high fat diet. The FGF19 transgenic mice had increased brown adipose tissue mass and decreased liver expression of acetyl coenzyme A carboxylase 2, providing two mechanisms by which FGF19 may increase energy expenditure. Consistent with the reduction in expression of acetyl CoA carboxylase 2, liver triglyceride levels were reduced.

19/AB/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013091764 BIOSIS NO.: 200100263603
fucosyltransferase1 and H-type complex carbohydrates modulate epithelial cell proliferation during prostatic branching morphogenesis
AUTHOR: Marker Paul C; Stephan Jean-Philippe; Lee James; Bald Laura; Mather Jennie P; Cunha Gerald R (Reprint)
AUTHOR ADDRESS: Department of Anatomy, University of California San Francisco, San Francisco, CA, 94143, USA**USA
JOURNAL: Developmental Biology 233 (1): p95-108 May 1, 2001 2001
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article

RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The prostate undergoes branching morphogenesis dependent on paracrine interactions between the prostatic epithelium and the urogenital mesenchyme. To identify cell-surface molecules that function in this process, monoclonal antibodies raised against epithelial cell-surface antigens were screened for antigen expression in the developing prostate and for their ability to alter development of prostates grown in serum-free organ culture. One antibody defined a unique expression pattern in the developing prostate and inhibited growth and ductal branching of cultured prostates by inhibiting epithelial cell proliferation. Expression cloning showed that this antibody binds fucosyltransferase1, an alpha-(1,2)-fucosyltransferase that synthesizes H-type structures on the complex carbohydrate modifications of some proteins and lipids. The lectin UEA I that binds H-type 2 carbohydrates also inhibited development of cultured prostates. These data demonstrate a previously unrecognized role for fucosyltransferase1 and H-type carbohydrates in controlling the spatial distribution of epithelial cell proliferation during prostatic branching morphogenesis. We also show that fucosyltransferase1 is expressed by epithelial cells derived from benign prostatic hyperplasia or prostate cancer; thus, fucosyltransferase1 may also contribute to pathological prostatic growth. These data further suggest that rare individuals who lack fucosyltransferase1 (Bombay phenotype) should be investigated for altered reproductive function and/or altered susceptibility to benign prostatic hyperplasia and prostate cancer.

19/AB/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012402664 BIOSIS NO.: 200000120977
Source, catabolism and role of the tetrapeptide N-Acetyl-Ser-Asp-Lys-Pro within the testis
AUTHOR: Stephan Jean-Philippe; Melaine Nathalie; Ezan Eric; Hakovirta Harri ; Maddocks Simon; Toppari Jorma; Garnier Danielle-Helene; Wdzieczak-Bakala Joanna; Jegou Bernard (Reprint)
AUTHOR ADDRESS: Groupe d'Etude de la Reproduction chez le Male (GERM), INSERM U-435, Universite de Rennes I, Campus de Beaulieu, 35042, Rennes, Bretagne, France**France
JOURNAL: Journal of Cell Science 113 (1): p113-121 Jan., 2000 2000
MEDIUM: print
ISSN: 0021-9533
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The tetrapeptide N-Acetyl-Seryl-Aspartyl-Lysyl-Proline (AcSDKP) is a natural regulator of hematopoietic stem cell proliferation. The present study was aimed at investigating the presence and the role of AcSDKP in rat testis. Specific immunoreactivity was always observed in the interstitial tissue at all stages of testicular development and in elongated spermatids at 45 days of age and in adults. In accordance with the interstitial labeling, high AcSDKP levels were detected in Leydig cell and testicular macrophage culture media and cell extracts, as well as in the testicular interstitial fluid (TIF). Much lower concentrations were found in peritubular cells and Sertoli cells cultures, whereas very low concentrations were present in cultured spermatocytes and spermatids.

In contrast to the slight degradation rate of AcSDKP observed in the spermatocyte and spermatid culture media, no catabolism of the peptide was seen in testicular somatic cell culture medium. Furthermore, the degradation rate of AcSDKP was much lower in TIF than in peripheral blood plasma. Despite the very strong evidence indicating that Leydig cells and testicular macrophages produce AcSDKP, the selective destruction of these cells did not result in any change in AcSDKP levels in TIF or in plasma. This suggests a compensatory mechanism ensuring constant levels of the peptide in TIF when interstitial cells are absent. Finally, in vitro, in the presence of AcSDKP, significantly more (3H)thymidine incorporation was found in A spermatogonia. In conclusion, this study establishes the presence of very high concentrations of AcSDKP in rat testis and demonstrates its Leydig cell and testicular macrophage origin. The presence of AcSDKP in the TIF and its stimulatory effect on thymidine incorporation in spermatogonia very strongly suggest its implication in the paracrine control of spermatogenesis.

19/AB/9 (Item 9 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012342648 BIOSIS NO.: 200000060961
Selective cloning of cell surface proteins involved in organ development: Epithelial glycoprotein is involved in normal epithelial differentiation
AUTHOR: Stephan Jean-Philippe (Reprint); Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Research Immunochemistry-AAT, Genentech, Inc., South San Francisco, CA, USA**USA
JOURNAL: Endocrinology 140 (12): p5841-5854 Dec., 1999 1999
MEDIUM: print
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Coordinating the activities of neighboring cells during development in multicellular organisms requires complex cellular interactions involving secreted, cell surface, and extracellular matrix components. Although most cloning efforts have concentrated on secreted molecules, recent work has emphasized the importance of membrane-bound molecules during development. To identify developmental genes, we raised antibodies to normal embryonic pancreatic epithelial cell surface proteins. These antibodies were characterized and used to clone the genes coding for the proteins by a panning strategy. Using this approach, we cloned the rat homologue of the mouse epithelial glycoprotein (EGP). Our immunohistochemistry data, describing the expression of EGP during rat development, as well as our in vitro data, looking at the effect of the anti-EGP antibody and the extracellular domain of EGP on embryonic pancreatic epithelial cell number and volume, strongly suggest a role for EGP during pancreatic development.

19/AB/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012209803 BIOSIS NO.: 199900469463
Distribution and function of the adhesion molecule BEN during rat development

AUTHOR: Stephan Jean-Philippe (Reprint); Bald Laura; Roberts Penelope E (Reprint); Lee James; Gu Qimin; Mather Jennie P (Reprint)
AUTHOR ADDRESS: Department of Protein Chemistry, Genentech, Inc., South San Francisco, CA, 94080-4990, USA**USA
JOURNAL: Developmental Biology 212 (2): p264-277 Aug. 15, 1999 1999
MEDIUM: print
ISSN: 0012-1606
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: It is well established that the notochord influences the development of adjacent neural and mesodermal tissue. Involvement of the notochord in the differentiation of the dorsal pancreas has been demonstrated. However, our knowledge of the signals involved in pancreatic development is still incomplete. In order to identify proteins potentially implicated during pancreatic differentiation, we raised and characterized monoclonal antibodies against previously established embryonic pancreatic ductal epithelial cell lines (BUD and RED). Using the MAb 2117, the cell surface antigen 2117 (Ag 2117) was cloned. The predicted sequence for Ag 2117 is the rat homologue of BEN. Initially reported as a protein expressed on epithelial cells of the chicken bursa of Fabricius, BEN is expressed in a variety of tissues during development and described as a marker for the developing central and peripheral chicken nervous systems. A role has been suggested for BEN in the adhesion of stem cells and progenitor cells to the blood-forming tissue microenvironment. In this study, we demonstrate that BEN, initially expressed exclusively in the notochord during the early development of rat, is implicated in pancreatic development. We show that Ag 2117 regulates the pancreatic epithelial cell growth through the ras and Jun kinase pathways. In addition, we demonstrate that Ag 2117 is able to regulate the expression of the transcription factor PDX1, required for insulin gene expression, in embryonic pancreas organ cultures.

19/AB/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011759356 BIOSIS NO.: 199900019016
Selective cloning of cell surface proteins involved in organ development: EGP is involved in normal epithelial differentiation
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qimin; Helmrich Angela; Barnes David; Devaux Brigitte; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p436A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

19/AB/12 (Item 12 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011756285 BIOSIS NO.: 199900015945

Distribution and function of the rat homologue of the adhesion molecule BEN during development
AUTHOR: Stephan Jean-Philippe; Roberts Penelope E; Bald Laura; Lee James; Gu Qumin; Devaux Brigitte; Mather Jennie P
AUTHOR ADDRESS: Dep. Protein Chem., Genentech Inc., South San Francisco, CA 94080-4990, USA**USA
JOURNAL: Molecular Biology of the Cell 9 (SUPPL.): p200A Nov., 1998 1998
MEDIUM: print
CONFERENCE/MEETING: 38th Annual Meeting of the American Society for Cell Biology San Francisco, California, USA December 12-16, 1998; 19981212
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

19/AB/13 (Item 13 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011325739 BIOSIS NO.: 199800119986
In vitro regulation of an inducible-type NO synthase in the rat seminiferous tubule cells
AUTHOR: Bauche Francoise (Reprint); Stephan Jean-Philippe; Touzalin Anne Marie; Jegou Bernard
AUTHOR ADDRESS: Groupe Etude Reproduction chez Male, INSERM U-435, Univ. Rennes I, Campus de Beaulieu, 35042 Rennes, Bretagne, France**France
JOURNAL: Biology of Reproduction 58 (2): p431-438 Feb., 1998 1998
MEDIUM: print
ISSN: 0006-3363
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Rat Sertoli cells express an inducible nitric oxide synthase isoform (iNOS) in response to the combined addition of the cytokines-interferon gamma (IFNgamma), tumor necrosis factor alpha (TNFalpha), interleukin-1alpha (IL-1alpha)-and lipopolysaccharides (LPS). We demonstrated that the addition of cytokines and lipopolysaccharides (C+L) to cultured peritubular cells resulted in high nitrite and iNOS mRNA levels, indicating the induction of an iNOS isoform. This enzyme was not induced in cultured pachytene spermatocytes or spermatids. Nitrite production in Sertoli cells and peritubular cells required both IFNgamma and TNFalpha and was potentiated by LPS, whereas IL-lot was ineffective. The induction of nitrite production and iNOS mRNA by IFNgamma+TNFalpha+LPS could be further enhanced by basic fibroblast growth factor in Sertoli cells but not in peritubular cells. In contrast, transforming growth factor beta markedly reduced this induction in peritubular cells but had no effect on Sertoli cells. FSH positively modulated the C+L-induced iNOS in Sertoli cells. Dibutyryl cAMP had a synergistic effect with C+L on NOS activity in both Sertoli cells and peritubular cells. In contrast, testosterone did not influence basal or induced NOS activity in these two cell types. These data show that NOS activity in the somatic cells of the seminiferous tubules is induced and regulated by multiple factors that act in combination, and suggest that nitric oxide may participate in the endocrine and paracrine control of testicular function.

19/AB/14 (Item 14 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011251715 BIOSIS NO.: 199800045962
Regulation of Sertoli cell IL-1 and IL-6 production in vitro
AUTHOR: Stephan Jean-Philippe; Syed Viqar; Jegou Bernard (Reprint)
AUTHOR ADDRESS: Groupe d'Etude Reproduction chez le Male, INSERM U-435, Universite Rennes 1, Campus de Beaulieu 35042 Rennes, Bretagne, France** France
JOURNAL: Molecular and Cellular Endocrinology 134 (2): p109-118 Nov. 15, 1997 1997
MEDIUM: print
ISSN: 0303-7207
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Interleukin-1 (IL-1) and IL-6 are pleiotropic cytokines produced by a large variety of cell types. In the testis, Sertoli cells produce IL-1alpha and IL-6. Previous studies have demonstrated that, in vitro, Sertoli cell IL-1alpha production is stimulated by some inducers of macrophage IL-1, as well as by phagocytosis of residual bodies. Furthermore, we have also shown that IL-1alpha is able to enhance Sertoli cell IL-6 production by an autocrine action. The aim of the present study was to further investigate the regulation of Sertoli cell IL-1 and IL-6 production. Three categories of potential regulators were tested; the lipopolysaccharide (LPS) and the yeast extract zymosan; follicle stimulating hormone (FSH), testosterone and dexamethasone; tumor necrosis factor alpha (TNFalpha), interferon gamma (IFNgamma) and the nerve growth factor beta (NGFbeta). It was found that zymosan (400-800 mug/ml) and LPS (20 mug/ml) stimulated Sertoli cell IL-1 and IL-6 production. FSH (1 X 10-2-1 mug/ml) and NGF (25-200 mug/ml) stimulated Sertoli cell IL-6 levels in a dose-dependent manner but had no effect on IL-1. The effect of testosterone on Sertoli cell IL-1 and IL-6 secretion was biphasic: dramatic increased secretion with low concentrations (0.01-1 nM) and no effect with the higher concentration tested (100 nM). Dexamethasone reduced LPS-induced IL-1 and IL-6 production in a concentration-responsive manner (0.04-0.4 and 0.4-40 ng/ml, respectively). Addition of TNFalpha to Sertoli cells resulted in a dose-dependent increase of both cytokines (50-100 U/ml for IL-1, 100-200 U/ml for IL-6). In the case of IFNgamma, intermediate concentrations (50-100 U/ml) stimulated IL-1alpha, whereas the highest concentrations (200-400 U/ml) inhibited IL-6. It is concluded that regulation of Sertoli cell IL-1 and IL-6 is very complex as it involves factors as different as hormones, paracrine factors and activators of macrophages. The latter agents may be mimicking the action of pathogens or the action of intratesticular agents whose nature remains to be elucidated.

19/AB/15 (Item 15 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0011227083 BIOSIS NO.: 199800021330
Characterization of cell surface proteins using antibodies raised to antigens from pancreatic cell lines
AUTHOR: Stephan Jean-Philippe; Bald Laura; Roberts Penny; Mather Jennie P
JOURNAL: Molecular Biology of the Cell 8 (SUPPL.): p328A Nov., 1997 1997
MEDIUM: print

CONFERENCE/MEETING: 37th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 13-17, 1997; 19971213
SPONSOR: American Society for Cell Biology
ISSN: 1059-1524
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Citation
LANGUAGE: English

19/AB/16 (Item 16 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009992790 BIOSIS NO.: 199598460623
Nitric oxide production by Sertoli cells in response to cytokines and lipopolysaccharide
AUTHOR: Stephan Jean-Philippe; Guillemois Cyrille; Jegou Bernard; Bauche Francoise (Reprint)
AUTHOR ADDRESS: Groupe d'Etude Reproduction chule Male, INSERM U.435, Univ. Rennes I, Campus Beaulieu, 35042 Rennes Cedex, Bretagne, France**France
JOURNAL: Biochemical and Biophysical Research Communications 213 (1): p 218-224 1995 1995
ISSN: 0006-291X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Nitric oxide (NO) is formed from L-arginine residues by nitric oxide synthase (NO Synthase) in many types of cells and acts as an intercellular messenger in several physiological systems. In the present study, we demonstrate that a combination (CL) of interleukin-1-alpha, interferon gamma, tumor necrosis factor alpha and lipopolysaccharide induces nitrite (NO-2-) production in cultured rat Sertoli cells. This biosynthesis of NO-2- requires a lag time period of 18 hr and then increases for at least 96 hr; it is prevented by two NO Synthase inhibitors, N-G-monomethyl-L-arginine and aminoguanidine. Northern blot analysis shows the induction of a macrophage-like NO Synthase mRNA synthesis in Sertoli cells cultured for a minimum of 6 hr in the presence of CL, with maximal levels after 12 to 30 hr of incubation. These results indicate for the first time that cultured rat Sertoli cells express an inducible NO Synthase isoform in response to a combination of cytokines and lipopolysaccharide.

19/AB/17 (Item 17 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009906406 BIOSIS NO.: 199598374239
Residual bodies activate Sertoli cell interleukin-1-alpha (IL-1-alpha) release, which triggers IL-6 production by an autocrine mechanism, through the lipoxygenase pathway
AUTHOR: Syed Viqar; Stephan Jean-Philippe; Gerard Nadine; Legrand Alain; Parvinen Martti; Bardin C Wayne; Jegou Bernard (Reprint)
AUTHOR ADDRESS: GERM-INSERM U-435, Univ. de Rennes 1, Campus de Beaulieu, 35042 Rennes Cedex, Bretagne, France**France
JOURNAL: Endocrinology 136 (7): p3070-3078 1995 1995
ISSN: 0013-7227
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Interleukin-1 (IL-1) and IL-6 are produced by Sertoli cells. As IL-1 stimulates IL-6 production in some tissues, the cascade of events that results in IL-6 secretion by Sertoli cells was studied. The addition of IL-1-alpha to Sertoli cells resulted in a time-dependent increase in IL-6 secretion. Incubation of Sertoli cells with two known stimulators of IL-1 production, lipopolysaccharide (LPS) and residual bodies, resulted in a significant increase in IL-1 release into the medium several hours before IL-6 release. That IL-1 is essential for IL-6 production from Sertoli cells was established by blocking the actions of LPS and residual bodies with an anti-IL-1-alpha antibody. An increase in the release of IL-1 before IL-6 was also observed in medium obtained from staged segments of intact seminiferous tubules; IL-1 reached a maximum level at stage VIII, when mature spermatozoa are released and residual bodies are formed and phagocytosed. The secretion of IL-6 was low during this stage and then increased progressively from stage IX onward, consistent with IL-1 stimulation of IL-6. The pathway of IL-1-alpha-induced release of IL-6 was studied in the presence of agents that influence arachidonic acid release and metabolism. IL-1-alpha was found to stimulate arachidonic acid release by Sertoli cells. Furthermore, a phospholipase A-2 inhibitor, aristolochic acid, significantly decreased IL-1-, LPS-, and pyrularia pubera thionin-induced IL-6 secretion from Sertoli cells. Indomethacin, a specific inhibitor of the cyclooxygenase pathway, had no significant effect on basal, but enhanced IL-1- and LPS-stimulated IL-6 production. The involvement of arachidonic acid metabolites produced in the lipoxygenase pathway on the release of IL-6 was investigated indirectly, using nordihydroguaiaretic acid. This inhibitor reduced basal and IL-1-alpha- and LPS-stimulated IL-6 production. Ethacrynic acid, an inhibitor of peptidoleukotriene synthesis, also reduced basal IL-6 levels and blocked IL-1-alpha- as well as LPS-induced IL-6 secretion. It is concluded that IL-1 produced by Sertoli cells in response to LPS or residual bodies induces IL-6 through the lipoxygenase pathway.

19/AB/18 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

15338114 PMID: 14976145

Fibroblast growth factor 19 increases metabolic rate and reverses dietary and leptin-deficient diabetes.

Fu Ling; John Linu M; Adams Sean H; Yu Xing Xian; Tomlinson Elizabeth; Renz Mark; Williams P Mickey; Soriano Robert; Corpuz Racquel; Moffat Barbara; Vandlen Richard; Simmons Laura; Foster Jessica; Stephan Jean-Philippe; Tsai Siao Ping; Stewart Timothy A

Genentech Inc., 1 DNA Way, South San Francisco, California 94080, USA.

Endocrinology (United States) Jun 2004, 145 (6) p2594-603, ISSN 0013-7227 Journal Code: 0375040

Publishing Model Print-Electronic; Comment in Endocrinology. 2004 Jun;145(6) 2591-3; Comment in PMID 15140837

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Hormonal control of metabolic rate can be important in regulating the imbalance between energy intake and expenditure that underlies the development of obesity. In mice fed a high-fat diet, human fibroblast growth factor 19 (FGF19) increased metabolic rate [1.53 +/- 0.06 liters O(2)/h.kg(0.75) (vehicle) vs. 1.93 +/- 0.05 liters O(2)/h.kg(0.75) (FGF19);

P < 0.001] and decreased respiratory quotient [0.82 +/- 0.01 (vehicle) vs. 0.80 +/- 0.01 (FGF19); P < 0.05]. In contrast to the vehicle-treated mice that gained weight (0.14 +/- 0.05 g/mouse.d), FGF19-treated mice lost weight (-0.13 +/- 0.03 g/mouse.d; P < 0.001) without a significant change in food intake. Furthermore, in addition to a reduction in weight gain, treatment with FGF19 prevented or reversed the diabetes that develops in mice made obese by genetic ablation of brown adipose tissue or genetic absence of leptin. To explore the mechanisms underlying the FGF19-mediated increase in metabolic rate, we profiled the FGF19-induced gene expression changes in the liver and brown fat. In brown adipose tissue, chronic exposure to FGF19 led to a gene expression profile that is consistent with activation of this tissue. We also found that FGF19 acutely increased liver expression of the leptin receptor (1.8-fold; P < 0.05) and decreased the expression of acetyl coenzyme A carboxylase 2 (0.6-fold; P < 0.05). The gene expression changes were consistent with the experimentally determined increase in fat oxidation and decrease in liver triglycerides. Thus, FGF19 is able to increase metabolic rate concurrently with an increase in fatty acid oxidation.

19/AB/19 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

14689529 PMID: 12626724
Nucleic acid capture assay, a new method for direct quantitation of nucleic acids.
Tsai Siao Ping; Wong Anne; Mai Elaine; Chan Pamela; Mausisa Grace; Vasser Mark; Jhurani Parkash; Jakobsen Mogens H; Wong Wai Lee T; Stephan Jean-Philippe
Assay and Automation Technology Department, Genentech, Inc., 1 DNA Way, South San Francisco, CA 94080, USA.
Nucleic acids research (England) Mar 15 2003, 31 (6) pe25, ISSN 1362-4962 Journal Code: 0411011
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Technologies allowing direct detection of specific RNA/DNA sequences occasionally serve as an alternative to amplification methods for gene expression studies. In these direct methods the hybridization of probes takes place in complex mixtures, thus specificity and sensitivity still limit the use of current technologies. To address these challenges, we developed a new technique called the nucleic acid capture assay, involving a direct multi-capture system. This approach combines a 3'-ethylene glycol scaffolding with the incorporation of 2'-methoxy deoxyribonucleotides in the capture sequences. In our design, all nucleotides other than those complementary to the target mRNA have been replaced by an inert linker, resulting in significant reductions in non-specific binding. We also provide a versatile method to detect the presence of captured targets by using specific labeled probes with alkaline phosphatase-conjugated anti-label antibodies. This direct, flexible and reliable technique for gene expression analysis is well suited for high-throughput screening and has potential for DNA microarray applications.
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6/AB/11 (Item 11 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0013516965 BIOSIS NO.: 200200110476
Human monoclonal antibody specifically binding to surface antigen of cancer cell membrane
AUTHOR: Hosokawa S; Tagawa T; Hirakawa Y; Ito N; Nagaike K
AUTHOR ADDRESS: Kawasaki, Japan**Japan
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1211 (3): p2963 June 16, 1998 1998
MEDIUM: print
PATENT NUMBER: US 5767246 PATENT DATE GRANTED: June 16, 1998 19980616
PATENT CLASSIFICATION: 530-388.8 PATENT ASSIGNEE: MITSUBISHI CHEMICAL CORPORATION PATENT COUNTRY: USA
ISSN: 0098-1133
DOCUMENT TYPE: Patent
RECORD TYPE: Citation
LANGUAGE: English

6/AB/18 (Item 18 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0012126649 BIOSIS NO.: 199900386309
Antibodies produced by mice immunized with recombinant vaccinia viruses expressing two different types of a major Theileria sergenti surface antigen (p32) react with the native surface antigen
AUTHOR: Takasima Yasuhiro; Xuan Xuenan; Matsumoto Yasunobu; Onuma Misao; Otsuka Haruki (Reprint)
AUTHOR ADDRESS: Department of Global Agricultural Science, Graduate School of Agricultural and Life Science, The University of Tokyo, Tokyo, Japan** Japan
JOURNAL: Veterinary Parasitology 84 (1-2): p65-73 July, 1999 1999
MEDIUM: print
ISSN: 0304-4017
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A 32 kDa major surface antigen, p32, of Theileria sergenti at the piroplasm stage is the main target of the host immune response. The immunogenic property of the p32 varies in some strains among the population of Theileria sergenti in Japan where the Chitose type and the Ikeda type are the most common varieties. We have constructed vaccinia virus recombinants vv/p32C and vv/p32I which harbor the Chitose and Ikeda types of p32 gene, respectively. It was found that vv/p32C and vv/p32I produced type-specific p32 which did not cross react with the monoclonal antibodies (mAbs) against the other type of p32. When mice were immunized with vv/p32C and vv/p32I, antibodies against p32 were detectable 2 weeks after the immunization, and these antibodies reacted with the native surface antigen in purified T. sergenti merozoite.

6/AB/32 (Item 32 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0009911078 BIOSIS NO.: 199598378911

Engineered anti-CD38 monoclonal antibodies for immunotherapy of multiple myeloma

AUTHOR: Ellis Jonathan H (Reprint); Barber Karen A; Tutt Alison; Hale Christine; Lewis Alan P; Glennie Martin J; Stevenson George T; Crowe J Scott
AUTHOR ADDRESS: Wellcome Foundation Ltd., Langley Court, South Eden Park Rd., Beckenham, Kent BR3 3BS, UK**UK

JOURNAL: Journal of Immunology 155 (2): p925-937 1995 1995
ISSN: 0022-1767
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Multiple myeloma is a malignancy of plasma cells for which there is no effective treatment. To develop an immunotherapeutic agent, we have raised a high affinity mAb (AT1 3/5) against CD38, one of the few well-characterized surface Ags present on myeloma cells. Since murine monoclonals have many disadvantages as human therapeutics, we prepared two engineered forms of the Ab: a CDR-grafted humanized IgG1 and a chimeric FabFc-2 (mouse Fab cross-linked to two human gamma-1 Fc). To retain affinity in the humanized Ab, a number of changes were required to the human framework regions of the heavy chain. In particular, through systematic mutagenesis and computer modeling, we identified a critical interaction between the side chains of residues 29 and 78, which may be important for the humanization of other Abs. The properties of the humanized IgG1 and FabFc-2 constructs were compared in a series of in vitro tests. Both constructs efficiently directed Ab-dependent cellular cytotoxicity against CD38-positive cell lines, but C was activated only poorly. Neither construct caused down-modulation of CD38, nor did they affect the NADase activity of CD38. Despite their differing structures, both Abs showed similar activity in most assays, although the humanized IgG1 was more potent at inducing monocyte cytotoxicity. These data represent the first direct comparison of CDR-grafted and chimeric FabFc-2 forms of the same Ab, and offer no support for the perceived advantages of the FabFc-2. These Abs show promise for therapy of multiple myeloma and other diseases involving CD38-positive cells.

6/AB/40 (Item 40 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

0009347992 BIOSIS NO.: 199497369277

Anti-apogens and anti-engulfens: Monoclonal antibodies reveal specific antigens on apoptotic and engulfment cells during chicken embryonic development

AUTHOR: Rotello Rocco J; Fernandez Pierre-Alain; Yuan Junying
AUTHOR ADDRESS: Cardiovascular Res. Cent., Mass. General Hosp., 149 13th St., Charlestown, MA 02129, USA**USA

JOURNAL: Development (Cambridge) 120 (6): p1421-1431 1994 1994
ISSN: 0950-1991
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have isolated a group of monoclonal antibodies that

specifically recognize either apoptotic or engulfment cells in the interdigit areas of chicken hind limb foot plates, and throughout the embryo. Ten of these antibodies (antiapogens) detect epitopes on dying cells that colocalize to areas of programmed cell death, characterized by the presence of apoptotic cells and bodies with typical cellular and nuclear morphology. Our results indicate that cells destined to die, or that are in the process of dying, express specific antigens that are not detectable in or on the surface of living cells. The detection of these apoptotic cell antigens in other areas of programmed cell death throughout the chick embryo indicates that different cell types, which form specific tissues and organs, may utilize similar cell death mechanisms. Six of the monoclonal antibodies (antiengulfens) define a class of engulfment cells which contain various numbers of apoptotic cells and/or apoptotic bodies in areas of programmed cell death. The immunostaining pattern of the anti-engulfen R15F is similar to that of an antibody against a common leukocyte antigen, suggesting the participation of cells from the immune system in the removal of apoptotic cell debris. These novel monoclonal antibody markers for apoptotic and engulfment cells will provide new tools to assist the further understanding of developmental programmed cell death in vertebrates.

6/AB/48 (Item 2 from file: 358)
DIALOG(R)File 358:Current BioTech Abs

080971 CBA Acc. No.: 14-05-004001 DOC. TYPE: Patent
Method for generation of antibodies to cell surface molecules.
AUTHOR: Boer, M. de; Conroy, L. B.
CORPORATE SOURCE: Cetus Oncology Corp., Emeryville, CA, USA
CODEN: USXXAM
PATENT NUMBER: US 5397703
PATENT APPLICATION: US 910222 (920709)
PUBLICATION DATE: 14 Mar 1995 (950314) LANGUAGE: English
ABSTRACT: A method is disclosed for generating an immortalized cell line which produces a monoclonal antibody specific against a selected membrane-associated antigen. It comprises: producing the membrane-associated antigen on the cell surface of insect cells; injecting the insect cells into a host animal; recovering from the host animal those cells which produce antibodies; immortalizing those cells for growth in cell culture; screening the immortalized cells for production of antibodies specific to the membrane associated antigen in a binding assay employing noninsect cells having the membrane-associated antigen on the surface of the noninsect cells; and selecting those immortalized cells which produce antibodies that bind to the membrane-associated antigen on the surface of the noninsect cells.
080971

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Set     Items   Description
S1    1987270   ANTIBOD?
S2     581541   MONOCLONAL
S3     553384   S1 AND S2
S4        359   "SURFACE ANTIGEN"
S5         54   S3 AND S4
S6         52   RD S5 (unique items)
S7          0   S3 AND "SERUM FREE"
S8          0   RL-65
S9         56   E3-E4
S10         0   RD S9
S11        51   RD S9 (unique items)
S12        10   E3-E4
S13        10   RD S12 (unique items)
S14        16   E1-E4
S15        13   RD S14 (unique items)
S16         0   E1AND E3
S17         0   AU='STEPHAN JEAN PHILIPPE' AND AU='STEPHAN JEAN-PHILIPPE'
S18        23   AU='STEPHAN JEAN PHILIPPE' OR AU='STEPHAN JEAN-PHILIPPE'
S19        19   RD S18 (unique items)
?
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ANTIBODY 170826
ANTIBODYS 34
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ELISAS 7689
FAC 3164
FACS 16690
FIBRONECTIN 14275
FIBRONECTINS 401
MONOCLONAL 90075
MONOCLONALS 2173
PREPARATION 1399027
PREPARATIONS 185301
PREPN 293063
PREPNS 8913
SERUM-FREE 15950
SERUM-FREES 0
SURFACE ANTIGEN N/A
18 561